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EDITORIAL

AMERICAN BOARD OF SURGERY ORGANIZED

IN answer to the demand for an agency which will attempt to certify competent surgeons the American Board of Surgery recently has been organized. This Board is a member of the Advisory Board of Medical Specialties which includes all of the boards of certification for the different medical specialties which have been already organized. Since boards were in existence for the certification of practitioners of some of the surgical specialties such as ophthalmology, otolaryngology, obstetrics and gynecology, genitourinary surgery and orthopedic surgery it is expected that the American Board of Surgery will be responsible for the certification of general surgeons as well as those practicing in the remaining specialized subdivisions of surgery.

Acting upon the invitation of the American Surgical Association the following surgical societies cooperated in the creation of the American Board of Surgery: the American Surgical Association, the Surgical Section of the American Medical Association, the American College of Surgeons, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association and the New England Surgical Society. The first three of these bodies which are national in scope have three representatives on the Board. All of the other societies have one representative each. The representatives of the cooperating societies are nominated by the society which they represent and upon approval of the Board shall become members of it. The term of membership on the Board will be six years.

Two groups of candidates are recognized for qualification by the Board.

(a) Those who have already amply demonstrated their fitness as trained specialists in surgery.

(b) Those who, having met the general and special requirements exacted by the Board, successfully pass its qualifying examination.

The first of these groups, the Founders Group, upon invitation by the Board will be chosen from the following:

(1) Professors and Associate Professors of Surgery in approved medical schools in the United States and Canada.

(2) Those who for fifteen years prior to the Board's organization have limited their practice to surgery.

(3) Members of the American Surgical Association, the Southern Surgical Association, the Western Surgical Association, the Pacific Coast Surgical Association and the New England Surgical Society, who are in good standing January 9, 1937.

All applications for the Founders Group must be received within two years of the Board's organization, January 9, 1937. No candidates for the Founders Group will be considered after that date.

Requirements for those to be qualified by examination will be as follows:

(1) Graduation from a medical school of the United States or Canada recognized by the Council on Medical Education and Hospitals of the A. M. A. or graduation from an approved foreign school.

(2) Completion of an internship of not less than one year in a hospital approved by the same Council, or its equivalent in the opinion of the Board.

(3) *Special Training.* A further period of graduate work of not less than three years devoted to surgery taken in a recognized graduate school of medicine or in a hospital or under the sponsorship accredited by the American Board of Surgery for the training of surgeons. This period of special training shall be of such character that the relation of the basic sciences of anatomy, physiology, pathology, bacteriology and biochemistry is emphasized. Knowledge of these sciences as applied to clinical surgery will be required in the examination. Adequate operative experience in which the candidate has assumed the whole responsibility will be required. An additional period of not less than two years of study or practice in surgery.

(4) The candidate must present to the Board sufficient evidence of good moral character as to justify it in the belief that he will not engage in fee splitting and other dishonest practices.

It is expected that the Board, with the assistance and cooperation of the American Medical Association and the American College of Surgeons, will be able to increase the facilities which now exist for the adequate training of young surgeons by means of residencies, fellowships, etc., in suitable hospitals.

The above requirements, especially those referring to surgical training, are subject to change from time to time as the existing opportunities for training in this field of specialization may be broadened.

The qualifying examination will be divided into two parts; Part I, written, and Part II, clinical, bedside and practical. The written part, Part I, will concern itself with general surgical problems and with the clinical application of the basic sciences of surgery to these problems. Part II, is entirely oral and will also concern itself, in the main, with general surgery and, as stated for Part I, clinical application of the basic sciences to the clinical problem represented. In addition to this, in Part II, an examination will be given to test the candidate's knowledge of operative surgery, *x*-ray plate interpretation and the principles and application of surgical anesthesia. Reexaminations will be allowed providing one year shall elapse between examinations.

This Board is a non-profit organization. All fees will be used, after a reasonable amount is set aside for necessary expenses in maintaining its office, conducting ex-

aminations, etc., to aid in improving existing opportunities for the training of the surgeon.

A certificate attesting to a candidate's qualifications in surgery after meeting the requirements of the Board will be issued, having been signed by its officers.

Any certificate issued by the Board shall be subject to revocation by the Board at any time in case it shall determine in its sole judgment, that a candidate, who has received a certificate, either was not properly qualified to receive it or has become disqualified since its receipt.

The Board will hold its first examination (Part 1, written) on September 20, 1937. All inquiries concerning applications for this examination should be received by the secretary's office promptly.

Requests for booklets of information, application blanks, and other information should be addressed to the Secretary, Dr. J. Stewart Rodman, 225 South 15th Street, Philadelphia, Pennsylvania.

Subscribers to The American Journal of Surgery *visiting New York City are invited to make the office of the publishers (The American Journal of Surgery, Inc., 49 West 45th Street, New York) their headquarters. Mail, packages or bundles may be addressed in our care. Hotel reservations will gladly be made for those advising us in advance; kindly notify us in detail as to requirements and prices.*

ORIGINAL ARTICLES

INFECTIONS OF THE SPINAL EPIDURAL SPACE: AN ASPECT OF VERTEBRAL OSTEOMYELITIS*

JEFFERSON BROWDER, M.D. AND RUSSELL MEYERS, M.D.

BROOKLYN, N. Y.

THOSE who have interested themselves with the surgical and pathological aspects of infections of the spinal epidural space have quite generally agreed in the belief that there are two modes by which septic agents may invade this space. In the first of these it is held that the infection extends directly from an inflammatory process in tissues adjacent to the vertebral column, such as decubitus ulcer, a carbuncle or a perinephritic abscess, into the fat and areolar tissue of the spinal epidural space. Such cases present no difficulty in our effort to understand their etiology and pathogenesis. In the second mode of invasion it is presumed that a septic metastasis arises from some more distant focus of infection, such as furuncle of the scalp or face, a diseased tonsil or a septic postabortional uterus, and by a hematogenous route establishes itself directly in the epidural space. The cases included under this heading constitute by far the larger group. Moreover, as will be indicated, they present certain features which run counter to the proposed interpretation of their pathogenesis. It is the purpose of the present paper to record our experiences with seven cases of spinal epidural infection and through a consideration of the symptomatology, clinical course and pathology of the disease to express a conviction concerning the pathogenesis of this second group, namely, that here is represented a complication of vertebral osteomyelitis. All instances of syphilitic and tuberculous lesions have been excluded from the present discussion.

CASE REPORTS

CASE 1. I. T., a thirty-three year old male piano mover, was admitted to the Kings County Hospital on April 1, 1929, complaining of pain in the back, abdominal distention and paralysis of the legs. The patient contracted a severe "cold" approximately six weeks before admission to the hospital. One week later he began to have dull aching pain in the upper lumbar region of the back, severe enough to confine him to bed. Within the next four or five days this persistent, agonizing pain began to radiate around "into the stomach" and down the left leg. By the end of the second week he was unable to urinate. The abdomen now became distended. On the day following the onset of the urinary retention, both legs became paralyzed and numb, and at this time the pain in the back and left leg abated, but the soreness across the upper abdomen persisted as before. The patient remained at home during the next three weeks. Incontinence of urine and feces developed, bedsores appeared and there was a high fever. He was brought to the hospital in a confused, semidelirious state.

Upon admission he was markedly dehydrated. Sloughing decubitus ulcers were present over the sacrum and both heels and there was dependent congestion of the skin. The temperature was 104 to 105°F., the pulse rate was 120 per minute and the respiratory rate was 40 per minute. His neck was rigid. There was complete flaccid paralysis, areflexia of the lower extremities and loss of cutaneous sensation up to the eighth thoracic dermatome. There was marked abdominal distention. The fundus of the urinary bladder could be palpated at the umbilicus and urine dribbled away constantly. The red blood cells numbered 5,376,000 per cu. mm., and the hemoglobin was 100 per cent. Blood sugar and creatinin were normal;

* From the Neurological Service of Bellevue Hospital, Foster Kennedy, M.D., Director, and the Neurosurgical Service of Kings County Hospital, Jefferson Browder, M.D., Director.

blood urea was 42 mgm. per 100 c.c. and the Wassermann (blood serum) was negative. A few pus cells were found in the urine. A spinal puncture was performed and, at a depth of 3 cm., in the third lumbar interspace, a drop of thick yellow pus was obtained. Smear and culture of this material revealed a non-hemolytic streptococcus and Staphylococcus aureus. The patient became rapidly weaker and died two days following admission.

Autopsy. The general autopsy findings were unimportant. Upon exposing the posterior spinous processes of the lumbar vertebrae, a large collection of yellow pus was encountered undermining the sacrospinous muscle of the left side. A small area of bony destruction was found in the left lamina of the third lumbar vertebra. This apparently had perforated both the internal and external cortices of the lamina (Fig. 1). The entire epidural space was filled with a creamy yellow pus which extended from the level of the second thoracic vertebra to the upper border of the fifth lumbar vertebra. When the dura was opened, inspection revealed no evidence of pathology of the leptomeninges or of the spinal cord. Microscopic study revealed congestion and exudative reaction of the pia mater, mild degenerative changes in the lateral columns of the spinal cord as high as the tenth thoracic segment, and a marked inflammatory reaction over the outer surface of the dura mater.

COMMENT. This case represents a typical instance of the so-called primary metastatic abscess in the spinal epidural space. The portal of entry for the bacteria probably was the throat. From the fact that the boring pain, so characteristic of bony infection, appeared one week after onset of the upper respiratory infection and some five days before the first neurological sign, it would seem reasonable to suppose that the osteomyelitis developed in the left lamina of the third lumbar vertebra as a metastasis from the pharynx. Subsequent to this, the suppurative process so established perforated the cortex of the lamina and invaded the spinal epidural space. This zone of osteomyelitis found at autopsy was small and did not involve the pedicle or the posterior spinous process of the involved vertebral arch. Indeed, its discovery was accidental rather than by design. It could easily have been overlooked, bathed, as it was, in a rather large lake of pus. It suggested strongly that many spinal epidural infections may be secondary to osteomyelitis of a part of a vertebra and not, as is most frequently maintained, metastatic infections primary in the spinal epidural space.

CASE II. M. B., a twenty-three year old housewife, was admitted to the Kings County Hospital on November 25, 1931, complaining of feverishness and pain in the back. Her past health had been excellent. Seven weeks before admission she underwent an interruption of a two months pregnancy by a midwife and during the next six weeks she suffered from pelvic pain, fever, chills and vaginal bleeding. At the end of this period, that is, November 18, 1931, she consulted a physician who "removed some material" from the uterus and opened a "gland" in the neck. Following this operation the vaginal bleeding ceased and there was abatement of pain. However the chills and fever continued. Three days later a dull gnawing pain made its appearance in the back. This was localized between the shoulder blades, was intensified by movement of the spine and became increasingly severe over a four day period, at which time the patient sought relief at the hospital.

Upon admission the temperature was 104°F., and the pulse rate was 130 per minute. The cervical glands were enlarged bilaterally and a seropurulent material was discharging from a small cutaneous opening just above the medial end of the right clavicle. Marked tenderness was present over the posterior spinous processes of the third, fourth and fifth thoracic vertebrae, but there was no palpable swelling here. The abdomen was moderately distended. There was a slight serosanguinous discharge from the uterus, but otherwise examination of the pelvis revealed nothing abnormal. The lower extremities could be moved voluntarily without difficulty and no gross sensory changes were discoverable. All reflexes were present and were considered to be within normal limits. There was a leucocytosis of 12,700 cells, of which 72 per cent were polymorphonuclear cells. The hemoglobin was 78 per cent. A few leucocytes were present in the urine. A blood culture taken at this time

was subsequently reported as negative. On the day following her admission to the hospital the patient noticed that the right lower extremity was "stiff." At this time she was unable to urinate.

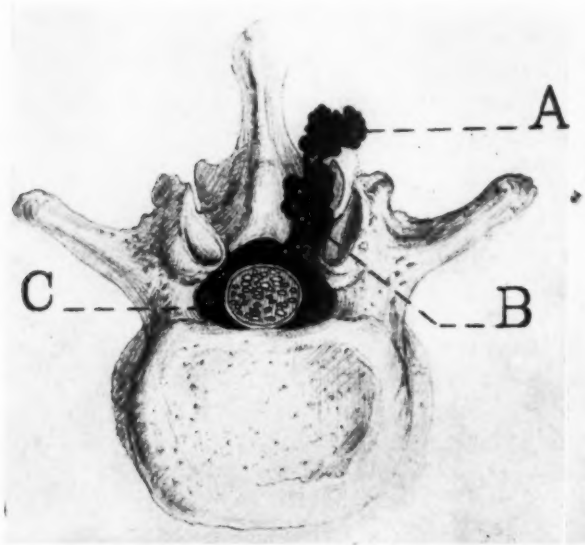


Fig. 1. Case I, patient I. T. Diagram illustrating an osteomyelitic process at B in the left lamina of the third lumbar vertebra and a rupture of the suppurative infection into the paravertebral tissues, A, and into the epidural space about the cauda equina, C.

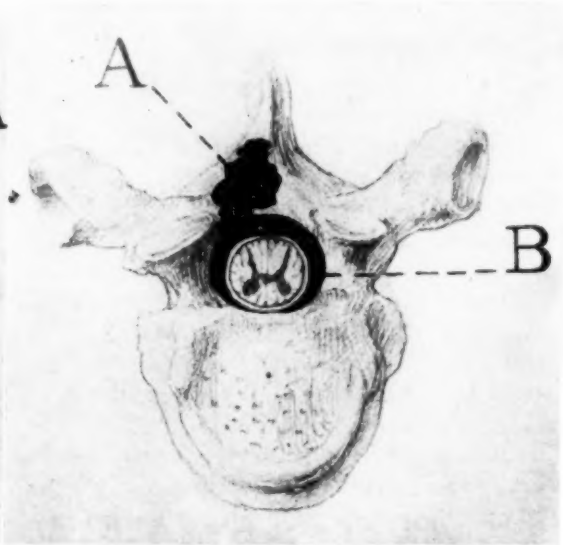


Fig. 2. Case II, patient M. B. Diagram illustrating an osteomyelitic process at A in the right lamina of the third thoracic vertebra with an extension through the inner bony cortex and into the spinal epidural space about the thoracic cord, B.

Examination revealed nuchal rigidity, tenderness along the lower cervical and upper thoracic spine, weakness of the right lower extremity and diminution of sensibility to pin prick over the left lower extremity. The tendon reflexes of the right lower extremity were more active than those of the left lower extremity. Twenty-four hours later both lower extremities had become completely paralysed and incontinence of urine and feces appeared.

A neurosurgical consultation was requested and the following significant findings were disclosed: rigidity of the neck; tenderness to pressure over the upper thoracic spine; abdominal distention; paralysis of the rectal and vesical sphincters; complete flaccid paralysis with deep and superficial areflexia from the costal margin downward; loss of all modalities of superficial sensitivity with preservation of deep sensation (including muscular pain on the left side) from the level of the umbilicus downward; hypesthesia, hypalgesia and thermhypesthesia on the right side from the level of the umbilicus downward; complete loss of superficial sensitivity over the right foot, lower third and lateral aspect of the leg on this side; preservation of acrognosis and vibratory sense in the right lower extremity and absence of response to plantar stimulation bilaterally. A lumbar puncture showed a xanthochromic spinal fluid, a pleocytosis of 200 cells per cu. mm. and evidence of complete block of the subarachnoid space as revealed by the Queckenstedt test. No organisms were found in the spinal fluid either by smear or culture. The Wassermann tests (blood serum and spinal fluid) were negative.

One week after her admission she was given a transfusion of 500 c.c. of whole blood. On December 4, 1931 a blood culture proved positive for both Staphylococcus aureus and a non-hemolytic streptococcus. A second transfusion was given December 6, 1931, and a blood culture taken at this time confirmed the previous findings. Meanwhile the abnormal neurological signs progressed and the sensory level rose as high as the sixth thoracic dermatome. The temperature varied in a spiking fashion between 97 and 105°F. On December 11, 1931, that is sixteen days following her admission, the patient became comatose and died.

Autopsy. At autopsy a general enlargement of the lymph glands was disclosed. There were multiple abscesses of the left kidney and an

advanced cystitis was evident from the sloughing of the mucosa of the bladder. The uterus did not appear enlarged and the endometrium was intact except for what appeared to be small, scattered petechial hemorrhages. Examination of the vertebral column disclosed an area of localized osteomyelitis involving the lamina on the right side of the third thoracic vertebra (Fig. 2). A collection of creamy pus occupied the entire epidural space between the level of the fifth cervical vertebra and that of the fourth lumbar vertebra. The spinal cord was removed. No visible pathological process was discoverable in the subdural and subarachnoid spaces. Inspection and palpation of the spinal cord revealed marked softening in the mid-thoracic region. When this zone was sectioned grossly, there was no demarcation discernible between the gray and white matter. Unfortunately, histological studies were not made.

COMMENT. It is quite evident that the experience with Case 1 did not leave a lasting impression. The symptom of severe pain in the spine preceding the onset of dysfunction of the spinal cord and presenting as it did, during the course of uterine infection, should have suggested the correct diagnosis. The clinical course of the patient's illness and the autopsy findings appear to indicate that the osteomyelitic involvement of the lamina antedated the infection of the spinal epidural space. Timely surgery might have saved this patient.

CASE III. J. L., a twenty-one year old baker, was admitted to the Long Island College Hospital on February 13, 1932, in a stuporous state. Thirteen days before admission the patient had contracted a "cough" and a sore throat. Four days after the onset there appeared a dull, throbbing, non-radiating pain in the back of the neck. Four days following the onset of the pain the patient found himself unable to raise the right lower extremity and within twelve hours the right hand became paralysed. On the day following the appearance of the paralysis of the right-sided extremities he became drowsy and gave evidence of mental aberrancy. The tenth day after the onset of the sore throat there was weakness of the left-sided extremities and urinary incontinence. When admitted to the hospital he was in a stuporous condition and could be aroused only by strong stimulation.

The skin of the entire body, as well as the sclerae, presented an icteric tint. The head was maintained in a fixed position, flexed laterally to the right. There was marked nuchal rigidity and forward flexion of the head elicited a painful response. The temperature was 99°F., pulse rate was 90 per minute and respiratory rate was 24 per minute. Pressure over the lateral aspects of the lower cervical spine produced such pain as to arouse the patient from his stupor. The neurological examination revealed anisocoria with a dilated fixed pupil on the right and a promptly reactive pupil on the left side. There was a complete flaccid paralysis of the right-sided extremities. The left lower extremity was paralyzed, but when dropped from the examiner's hand, it proved to possess a slight tonus. The left upper extremity was markedly paretic although a few voluntary movements were made with it. The thorax was immobile and respirations were entirely diaphragmatic in type. There was loss of the biceps brachii and of the radial periosteal reflexes on the right side. The pectoral and triceps jerks could be elicited on both sides. The abdominal and cremasteric reflexes were absent bilaterally and the deep reflexes of the lower extremities were likewise absent. Plantar stimulation elicited a flexor-type response on both sides. The bladder and rectal sphincters were paralysed. The sensory examination was somewhat contradictory owing to the patient's mental status. A blood count revealed 15,000 white cells per cu. mm. of which 78 per cent were of the polymorphonuclear type. The hemoglobin was 88 per cent. A trace of albumin and a few granular casts were found in the urine. The blood chemistry analysis was as follows: glucose 135 mgm. per 100 c.c., creatinin 1.5 mgm. per 100 c.c., urea 107 mgm. per 100 c.c. and uric acid 4 mgm. per 100 c.c. A lumbar puncture yielded xanthochromic spinal fluid under 20 mm. Hg. pressure. Queckenstedt's test demonstrated a partial subarachnoid block. There was a pleocytosis of 1350 mononuclear cells per cu. mm. in the spinal fluid. A Wassermann test (spinal fluid) was anticomplementary and the colloidal gold curve was 2355544333.

With this data at hand, the impression was that this was the terminal phase of a low cervical spinal epidural abscess complicating a septicemia. Owing to the respiratory embar-

rassment, the patient was placed in a Drinker respirator, but in spite of these efforts he died nine hours after admission.

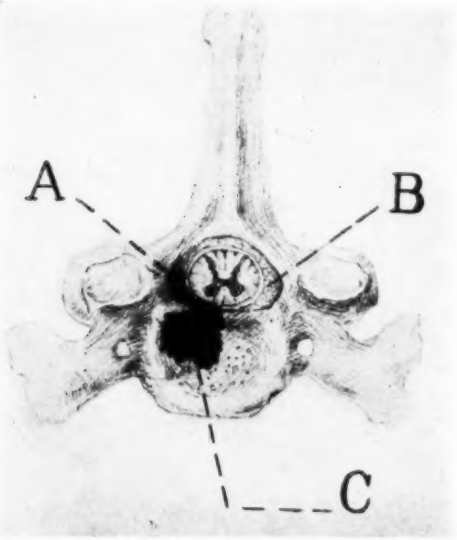


Fig. 3. Case III, patient J. K. Diagram illustrating an osteomyelitic destructive process in the posterior portion of the body of the seventh cervical vertebra, C. The pus has broken through the cortical bone and has established an abscess in the right anterolateral spinal epidural space, A, and a marked inflammatory reaction in the spinal epidural fat, B.

Autopsy. A moderate degree of general glandular enlargement was disclosed. The right tonsil was removed for culture. The spleen was moderately enlarged and was the seat of multiple septic infarcts. Similar infarcts were scattered throughout both lungs. The sulci and cisterns of the brain contained small amounts of thin turbid fluid, and one area of fibrinous exudate was found over the posterior aspect of the left cerebellar hemisphere. The vertebral column was exposed and an inflammatory destruction of bone was found in the right posterior portion of the body of the seventh cervical vertebra (Fig. 3). The spinal dura mater presented a marked congestion of its external surface from the level of the middle cervical region to that of the third thoracic vertebra. Marked edema was apparent about the seventh and eighth cervical and the first and second thoracic spinal nerve roots on the right side. The spinal cord was removed intact with its meninges. Inspection of the vertebral canal revealed an oval shaped abscess situated between the dura mater and the posterior longitudinal spinous ligament, extending from the upper part of the seventh cervical vertebra down as far as the midpart of the second thoracic vertebra. The vertebral column presented an inflammatory destruction of the bone in the right posterior portion of the body of the seventh cervical vertebra.

Following twenty-four hours of formalin fixation the cord and its meninges were sectioned. The gross appearance revealed thickening of the dura mater over the area corresponding to the abscess. A grayish cellular substance occupied the subdural space of the same levels. From the sixth cervical segment down to the second thoracic segment the spinal cord was soft, the gray and white matter being indistinguishable.

Microscopic sections at the level of the abscess showed marked thickening of both the dura mater and the arachnoid with a thin subdural collection of pus. The spinal cord itself was edematous and degeneration in the regions of the long columns was apparent. Many blood vessels of the cord were thrombosed, being filled with septic thrombi. No unusual cellular infiltration of the cord was noted. There was a moderate dilatation of the central canal. Cultures were made of the tonsils, lungs, spleen and epidural abscess. All of these foci yielded the Staphylococcus areus.

COMMENT. It is distinctly unusual, as indicated by a review of the literature on this subject, to find a spinal epidural abscess situated in the anterior aspect of the vertebral canal. Anatomical consideration accounts for this: The dura mater is attached along its anterior face to the posterior longitudinal spinous ligament so that no true space exists here as it does over the lateral and posterior aspects of the spinal dura. Nevertheless, as is well illustrated in this case and in Case VI of this series, an anterior abscess *may* occur and the associated spinal cord changes may be identical with those encountered in connection with infections limited to the dorsal or lateral aspects of the epidural space. We have been able to find four other cases in the literature in which anterior abscesses were described, one each by Mills and Spiller[81] and Mintzman[82] and two reported by Mixter and Smithwick.[84]

Case IV. E. L., a thirty-three year old seaman, was admitted to the Long Island College Hospital on December 16, 1931, complaining of severe pain in the upper part of the back just to the left of the spine. The past history indicated the contraction of a syphilitic infection four years prior to the present illness. He was treated for this over a period of three years and was then discharged by his physician as cured. Seven months prior to the onset of the present illness the patient suffered a crop of furuncles over the arms and legs. He recovered from these skin lesions completely. Three days before admission he was seized with a sharp pain, well localized alongside the spine, "high up in the back" and medial to the left scapula. The pain grew steadily in intensity and was unbearably severe whenever the slightest movement of the spine was made.

The only unusual physical finding at this time was tenderness along the left side of the upper thoracic spine. The temperature was 102°F. and the pulse rate was 90 per minute. The white blood cells numbered 20,400 per cu. mm. Roentgen ray examination of the thorax and spine was negative. Four days after admission a pleural friction rub was heard at the angle of the left scapula. Roentgen ray examination at this time disclosed a circumscribed shadow to the left of the spinal column at the level of the fifth and sixth ribs (Fig. 4), interpreted as an intrapleural collection of fluid. On the following day the patient complained of pain around the midpart of the left chest and of stiffness of the neck. Spinal puncture revealed a turbid fluid containing 1010 mononuclear cells per cu. mm. The total protein content of the spinal fluid was 52 mgm. per 100 c.c. There was no evidence of subarachnoid block. The Wassermann test (spinal fluid) was reported four plus.

The patient continued very ill for another week, at the end of which time the temperature began to "spike" between 102° and 103°F. with morning remissions. The blood count and the lumbar puncture were repeated on the nineteenth day following admission. The spinal fluid was turbid and contained 304 cells per cu. mm. There was a leucocytosis of the blood of 12,600 cells per cu. mm.

On the twenty-fourth day of hospitalization a visible and palpable swelling made its appearance about the upper thoracic spine, more prominent on the left side of the dorsal processes. Neurological examination revealed equal but very active tendon reflexes in both lower extremities and bilateral Babinski's sign. It was decided to explore the upper thoracic spine surgically. Upon exposing the posterior spinous processes of the third to the sixth thoracic vertebrae, the musculature was found to be honeycombed with abscesses. A considerable quantity of pus welled up on the left side between the transverse processes of the fifth and sixth thoracic vertebrae. Culture of the pus revealed Staphylococcus aureus. The drainage of this area seemed adequate, therefore the laminae were not removed. The entire area was packed with gauze and left open.

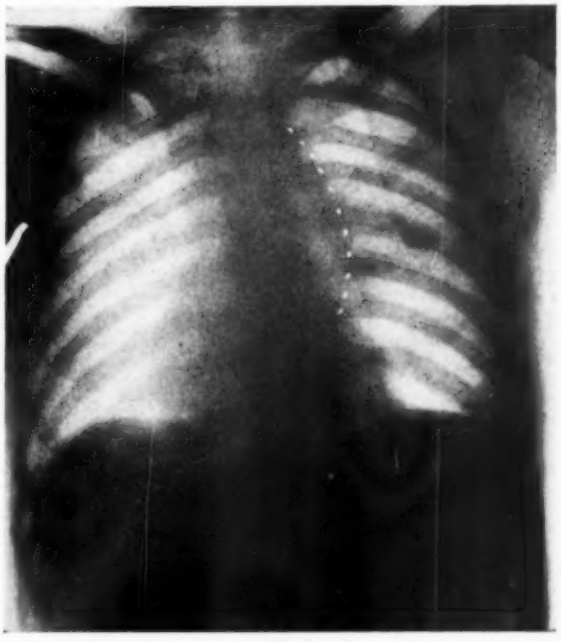


Fig. 4. Case IV, patient E. L. Roentgen ray study illustrating a circumscribed shadow (retouched with white line for purpose of delineation) to the left of the spinal column at the level of the fifth and sixth ribs. This shadow was later shown to correspond to an extrapleural infection. The latter was, in all probability, the extravertebral counterpart of a spinal epidural infection.

For the next two weeks the patient had a stormy course. Two transfusions of whole blood were required. Toward the end of this period however, the temperature subsided, the wound granulated and a general improvement was established so that on the forty-sixth postoperative day he was permitted to get out of bed. The physical signs in the chest had disappeared. Neurological examination now disclosed absent abdominal reflexes and hyperreflexia of the tendon responses of the right lower extremity. The Babinski sign was nega-

tive. On the sixty-first day following the operation a spinal puncture was performed. There was a complete subarachnoid block. The fluid was xanthochromic, clotted on standing and contained 100 mgm. of protein per 100 c.c.

Three months after operation, at a time when there was marked improvement in both the local and general conditions, the patient complained of a swelling in the region of the sixth rib in the left axillary line. A roentgenographic study at this time revealed a moderate thickening of the entire sixth rib which was interpreted as a low grade osteomyelitis. During the next two months the swelling subsided, the operative wound of the spine healed and the abnormal neurological signs disappeared. A spinal puncture was performed on the day of discharge. The fluid was clear, there was no evidence of spinal subarachnoid obstruction and the total protein content was 26 mgm. per 100 c.c.

COMMENT. The review of this case indicates that operation was deferred far too long. The probable sequence of events seems to have been: (1) osteomyelitis of the head of the left sixth rib; (2) a spinal epidural infection and an extrapleural abscess. It is interesting to note that a spinal subarachnoid block persisted for as long as two months after the abscess had been drained, yet at the time this was demonstrable the patient was walking about in a fairly normal manner.

CASE V. A. P., a thirteen year old schoolgirl, was admitted to the Kings County Hospital on November 12, 1931, complaining of paralysis of the lower extremities and urinary incontinence. Her past health had been good. The present illness began just one month before admission, when the upper part of her back was accidentally traumatized at play. There was some immediate discomfort but the child felt entirely well the following morning and remained so for the next three days. On the third day following the accident a dull aching pain appeared in the upper part of the back which radiated around the left side of the chest. The family doctor had a roentgen ray study made and this was said to have disclosed two fractured ribs. The chest was strapped with adhesive plaster but no relief was obtained. The boring pain in the back continued with increasing intensity until the twenty-second day following the trauma, at which time a sensation of "pins and needles" was experienced in both feet. On the day after the appearance of these parasthesiae, the lower extremities became paralyzed and felt numb. There was also a loss of sphincter control.

Within the next few days she was taken to the Queensboro Hospital where examination disclosed a complete flaccid paralysis and areflexia of the lower extremities. There was analgesia, thermanesthesia and hypesthesia from the sixth thoracic dermatome downward. The deep modalities of sensation, however, were preserved over these parts of the body. A pressure sore was present over the sacrum and there were hot water bag burns over the lateral aspects of both legs. Spinal puncture yielded xanthochromic fluid containing 50 mononuclear cells per cu. mm. No bacteria were found on smear or culture of the spinal fluid.

A month after the onset of her illness she was transferred to Kings County Hospital. She appeared acutely ill with a fever of 102°F. and a pulse rate of 110 per minute. The red blood cells were 3,200,000 per cu. mm., and the hemoglobin was 64 per cent. There were 5400 white blood cells per cu. mm. The urine contained many pus cells. Neurological examination revealed paralysis and areflexia of the lower extremities. A zone of hyperesthesia was present at the level of the cutaneous zone supplied by the seventh thoracic segment. Below this level deep and superficial modalities of sensation were lost, except for tactile sensibility over indefinite areas on the inner aspects of both thighs. The urinary bladder was distended and overflow incontinence was present. Spinal puncture showed a xanthochromic fluid containing 84 mononuclear cells per cu. mm. Bilateral jugular compression demonstrated a block of the spinal subarachnoid space. The Wassermann tests (spinal fluid and blood serum) were negative. The colloidal gold curve read 1122343200. Roentgenographic studies of the spine and ribs revealed no evidence of bony pathology.

It was deemed wise to defer operation until the urinary tract infection could be brought under control, especially since the neurological status appeared to be essentially stationary. After ten days the temperature was within normal limits and operation was performed under general anesthesia. The laminae were removed from the second, third and fourth dorsal vertebrae. A vascular inflammatory mass

was thus exposed, dark red in color and covering the entire dorsal aspect of the dura mater. It was necessary to rongeur away the laminae of the first dorsal vertebra in order to visualize the upper border of the mass. An attempt was made to remove the mass and the dura mater was accidentally torn. The entire subdural space at this level appeared to be filled with a soft grayish collection of cellular debris (Fig. 5). It was apparent that the inflammatory tissue could not be removed without seriously damaging the dura, so the mass was divided in its longitudinal axis and portions of the tissue were taken for histological study. Closure of the wound was made with catgut and a drain was left in place. The postoperative course was not remarkable except that a slight infection of the wound developed at the site of the drain.

The specimen consisted of many pieces of inflammatory connective tissue with scattered spots of necrosis. Other fragments were of a dry, grayish color, having a caseous appearance. The firm pieces were 4 to 5 mm. in thickness. Microscopically, the sections showed a conglomerate mass of tissue consisting of lymphocytes, plasma cells, large mononuclear phagocytes, young blood vessels and scattered polymorphonuclear leucocytes. Fibroblastic tissue was evident in some areas and in others there were zones of necrosis.

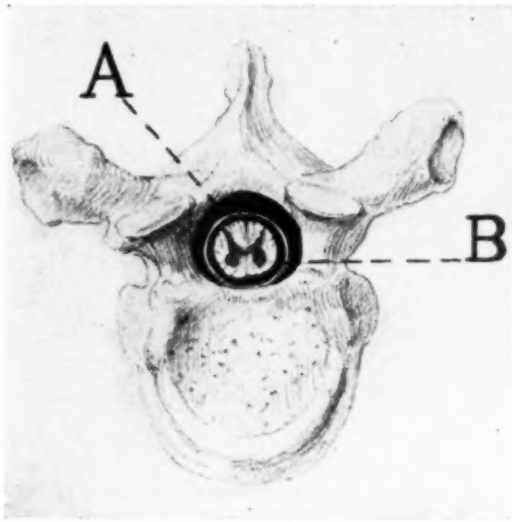


Fig. 5. Case v, patient A. P. Diagram illustrating the extent of the spinal epidural granuloma, A, together with the corresponding subdural inflammatory reaction encountered at operation, B.

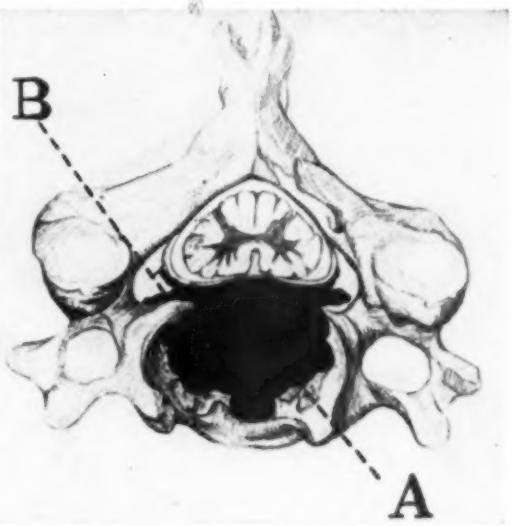


Fig. 6. Case vi, patient F. S. Diagram illustrating the extent of the anterior collection of pus in the vertebral canal, B, and the zone of osteomyelitis in the posterior part of the body of the fifth thoracic vertebra, A.

During the next two months there was a slow return of tactile sensibility. Pin prick, heat and cold could be appreciated, but this appreciation remained diminished as compared to normal parts. At times the desire to urinate was felt but the patient remained incontinent of urine. She was able to move the right great toe slightly. A lumbar puncture performed at this time indicated that there was no spinal subarachnoid block. The fluid was clear, contained only 6 cells per cu. mm. and had a total protein content of 25 mgm. per 100 c.c. The patient was transferred to a convalescent home on April 10, 1932, four and one-half months after operation. Examination in March, 1935 showed a well developed and well nourished young woman without complaint. She had largely regained control of the urinary bladder, although she occasionally "wet the bed." The gait remained slightly spastic.

COMMENT. This story illustrates the clinical course and findings of the type of case designated as spinal epidural granuloma. This lesion might be considered as a low grade infection limited to the epidural space and not secondary to osteomyelitis of a part of an adjacent vertebra. However, in spite of failure to uncover an area of bony destruction at operation,

the complaint of boring pain in a localized region of the back is similar in every respect to that encountered in other cases of this series in which vertebral osteomyelitis was proved. The character of the pain, moreover, is not unlike that associated with osteomyelitis in other bones of the body.

Case VI. F. S. a forty-nine year old white Belgian male, was admitted to Bellevue Hospital on February 25, 1929, with the chief complaint of painful respiration of five days duration. Of interest in the past history of the patient was the contraction of syphilis twenty-five years before admission. His wife had twelve pregnancies; nine children died in infancy of causes unknown to the patient and three children were living and well. The present illness began with a "head and chest cold" about two weeks before admission. At the end of a week the patient was profoundly ill, had a severe chill, vomited and coughed frequently. The sputum was blood tinged. Five days before admission a pain was experienced in the left upper chest and in the left shoulder.

On admission the patient was moderately dyspneic and cyanotic. The temperature was 102.8°F., the pulse rate was 100 per minute and the respiratory rate was 24 to 28 per minute. The pharynx was injected and the tongue coated. Percussion of the chest revealed slight dullness over the left upper chest posteriorly and auscultation here indicated a diminution of breath sounds. There was slight abdominal distention and tympany. Urinalysis was negative. There was a leucocytosis of 16,400 cells per cu. mm. of which 88 per cent were polymorphonuclear cells. The sputum revealed gram positive diplococci which were reported by the bacteriologist as pneumococci, type unclassified. Eight days after admission evidence of a collection of fluid in the left costophrenic sinus was obtained by physical examination and fluoroscopy.

On the tenth day following admission the first neurological sign was noted, namely, pain in the left hand and wrist and the inability to extend the fingers of this hand. Within four days, the interosseous muscles of the left hand appeared slightly atrophic and the right grip became weak. The neck was somewhat stiff. At this time the pneumonia was thought to be resolving satisfactorily and roentgenographic examination of the chest revealed evidence of a thickened pleura only. The white blood cells were now 21,000 per cu. mm. A neurological examination revealed weakness of the trapezius and sternomastoid muscles bilaterally, hyperactivity of the biceps reflexes of both upper extremities but more marked on the right, areflexia of the triceps and radial periosteal reflexes and hypotonicity and tenderness to palpation of the muscles of the upper extremities.

On the seventeenth day following admission, the patient was still profoundly ill, sweaty and feverish. He had complained almost daily of constant pain in the back of the neck. Radiographic examination of the cervical spine was negative.

On the twenty-second day after admission a neurological examination revealed weakness of the upper extremities as previously described. In addition, there were demonstrable sensory diminution and dysesthesiae over the ulnar side of the left forearm and hand and absence of the abdominal reflexes. There was moderate hyperreflexia of the knee and ankle jerks bilaterally and a bilateral Babinski sign. However, no sensory level was discoverable and the bladder function was apparently normal. The following day a lumbar puncture revealed a clear, canary-colored spinal fluid under normal pressure. The Queckenstedt test showed no evidence of spinal subarachnoid block. There were 40 lymphocytes per cu. mm. in the spinal fluid. The Wassermann test (spinal fluid) was negative and the colloidal gold curve read 0012211000.

On the twenty-eighth day after admission a definite sensory and motor level had appeared at the sixth cervical spinal segment and both bladder and rectal incontinence were present. An exploratory laminectomy was now performed from the level of the fifth cervical to the first thoracic vertebrae, inclusive. The bone itself was reported "suffering from inflammatory changes in that it is softer and mushier than normal." There was no undue bony angulation. The dura was "much inflamed, broadened, under increased tension and without pulsation." The pia mater was thick, opaque and adherent to the dura and the spinal cord by many fine adhesions. The spinal cord itself was greatly inflamed and presented an increase in the number and size of its vessels. Opposite

the seventh cervical vertebra the cord was wider than normal and gave the operator an impression that it was pushed backward, as if by a mass lying beneath the ventral surface of the dura mater. "It was felt that the patient was suffering from a diffuse inflammatory process, rather than from a localized pyomyelitis" and the dura mater was accordingly left open, the skin, muscles and fasciae being closed in layers. The wound healed well during the next ten days. The patient, however, remained febrile and complained of severe pain in the arms and legs. On the sixteenth postoperative day he gave evidence of early respiratory paralysis and died four days later.

Autopsy. The pleura showed evidence of acute and chronic pleuritis and there was an organizing lobular pneumonia of both lower lobes of the lungs. Both cystitis and ureteritis were present. There were decubitus ulcers of both heels and of the sacral region. The brain showed a softening of three-fourths of the right cerebellar hemisphere. That part of the spinal cord corresponding to segments c4, 5 and 6 appeared to be softened and light brown in color. Here the cord seemed to be impinged upon (as noted at the operation) so that it was displaced posteriorly. When it was pulled aside, a 1 cm. mass presented, representing a part of an osteomyelitic process involving practically the whole of the body of the fifth cervical vertebra (Fig. 6). An extradural collection of thin pus was found against the anterior face of the dura mater. Pus could be expressed from the softened vertebra. This had obviously escaped detection at operation. A smear of the pus revealed a pneumococcic organism. Unfortunately, culture and typing were not carried out. Histological study of the spinal cord revealed a "pachymeningitis hemorrhagica in the lower cervical region with complete softening and transverse myelitis of the lower cervical and upper dorsal segments."

COMMENTS. The noteworthy points brought out by this case are the following: (1) the offending organism was a pneumococcus, relatively rarely encountered in spinal epidural abscesses; (2) the abscess occupied an anterior postion, being in this respect also uncommon; (3) the abscess and osteomyelitis were overlooked at operation. This case emphasized a fact which has impressed itself upon us, both in our own experience and in our review of the literature, namely, that the failure to find osteomyelitis at operation or at necropsy does not warrant the assertion that it is not there. Had this patient been taken from the hospital without a postmortem examination, the case would have stood on the records as one of essential intrinsic inflammatory disease of the spinal cord.

CASE VII. F. O., a fifty-eight year old white male, was readmitted February 28, 1935, to Bellevue Hospital with a chief complaint of moderate but increasing weakness of both legs for a period of a year. The significant history goes back to December 31, 1922, at which time an abscessed tooth was extracted. The following day, the patient contracted a cold which required him to go to bed where he remained for nine weeks. During the first week of this illness he suffered headaches associated with a stiff neck and pain along the entire spine. At the end of the first week an abscess appeared on the left heel. This ruptured spontaneously and required four weeks to heal. The illness was diagnosed as "rheumatic fever." After recovering from the acute phase of his "fever," the patient complained of occasional attacks of severe spinal pain at the level of the scapulae. Three months later he noticed a weakness of the legs upon climbing and descending stairs. This weakness was slowly progressive for a period of a year, at which time stiffness and numbness had advanced to such a degree that a cane was required for walking. During this period he had inconstant pains in the back at various levels. The pains were apt to be more severe in the thoracic zone and were intensified by coughing. Six months after the onset there was a band-like sensation about the lower abdomen. This was not painful but "felt like a tight strap." There was unusual constipation but no urinary difficulty and no paresthesiae. For these complaints the patient sought relief and was admitted to Bellevue Hospital on June 15, 1925.

At that time he was a well developed and well nourished, forty-nine year old male, with a spastic gait and hyperreflexia of the lower extremities, more marked on the left. Active movements in the lower extremities were weak and passive movements were met with moderate involuntary resistance. Ankle clonus and

the Babinski phenomenon were present bilaterally. Sensory examination of the lower extremities revealed diminution of superficial sensitivity in all modalities. Acrognosis was preserved, but vibratory sense was diminished. There were two vague sensory levels, an upper one at dermatomes D7 and 8 below which there was gradual diminution in sensory appreciation down as far as the lower sensory level which began at D10. From here downward the sensory defect was more marked. The blood counts, hemoglobin and blood smears were normal. The color index was 0.6. The Wassermann test (blood serum) was negative, the urinalysis was normal and the blood pressure 120/80. A lumbar puncture was performed and yielded a clear, colorless spinal fluid under initial pressure of 80 mm. H_2O. Light and deep bilateral jugular compression produced no rise in this pressure. Voluntary straining produced prompt rise to 280 mm. H_2O. The globulin content of the spinal fluid was normal. There were 6 lymphocytes per cu. mm. The Wassermann test (spinal fluid) was negative and the colloidal gold curve read 0000012100. Roentgen ray examination of the entire spine was made and revealed no productive or destructive changes of the vertebrae.

A diagnosis of spinal extramedullary neoplasm was made and an exploratory laminectomy performed. The laminae of the second to the seventh dorsal vertebrae inclusive were removed. The following is the report of the important operative findings: "After removal of the posterior spinous processes, the laminae were found to be considerably thinned out and were in close apposition to firm underlying tissue. The lesion in the vertebral canal was a long, diffuse extradural process extending from D2 to D7 and it had the appearance of an old chronic inflammatory lesion. It occupied for the most part a position posterior to the spinal cord, but extended also laterally and anteriorly and could not be separated from the dura except by sharp dissection. It was excised with the dura attached for a distance of about 3½ inches. In a single cross section through this tissue there were two distinct foci, each consisting of a small pocket of pus and surrounded by granulation tissue. The dura had already been opened before the nature of the lesion was discovered. In view of this, it is feared that the patient may develop meningitis. The wound was closed in layers without drainage." The tissue was examined microscopically and was described as "a chronic productive inflammatory lesion which, in places, is distinctly suggestive of that encountered in Hodgkin's disease."

Following operation there was a progressive improvement. The wound healed well, vibratory sensibility returned within a week and the Babinski signs disappeared. Twelve days after operation a large pus pocket at the angle of the operative incision evacuated itself of three ounces of pus. The area was opened widely and dakenized hourly. A smear of this pus revealed "gram negative intracellular biscuit-shaped diplococci." A second abscess cavity was opened following the first evacuation. This, too, contained gram negative intracellular diplococci. Motor power steadily returned and the hyperreflexia and the sensory levels disappeared. Two and one-half months after operation the patient was discharged, neurologically recovered.

He remained well in all respects until the midwinter of 1934, that is, for eight and one-half years after his discharge. At this time he noted a tired feeling in his legs toward the end of his day's work and weakness in descending stairs. This weakness progressed steadily for the period of a year and during the month of January, 1935, became more marked. There were no paresthesiae or girdle sensations. He was readmitted to Bellevue Hospital on February 28, 1935, and the following signs were found: slight paresis of the lower extremities with a slightly dragging gait, hyperactive deep reflexes of the right side, absence of the abdominal reflexes and sluggishness of the right cremasteric reflex, hypesthesia below dermatome D4, and loss of vibratory sensibility below the iliac crests. Acrognosis was normal and there was no extensor toe response. The temperature, pulse and respiration rates were normal. Blood pressure was 140/85 and the urinary and blood studies were normal. A Queckenstedt test gave evidence of a partial block and the spinal fluid pressure dropped from 110 mm. H_2O to zero levels following the removal of 20 c.c. of cerebrospinal fluid. The fluid was yellowish and contained 2 lymphocytes per cu. mm. Its protein content was 95 mgm. per 100 c.c. The Wassermann test (spinal fluid) was negative and the colloidal gold curve was 2443432100. Roentgenographic studies of the lungs were negative, those of the upper thoracic spine revealed an extensive zone

of density in the area of bony defect resulting from the previous laminectomy.

The old laminectomy wound was reopened two weeks after the patient's second admission and an extradural inflammatory process was exposed. The dura was much thickened and covered with tough granulation tissue. Several small cavities of pus buried in this tissue were opened. The stump of the lamina on the left side of the third thoracic vertebra was the seat of a deeply excavating osteomyelitic process (Fig. 7). A culture of the pus was taken and was later reported positive for Staphylococcus aureus and a gram negative bacillus. The zone of osteomyelitis was rongeured away and the entire wound was left open.

For the first few postoperative days urinary incontinence and dysuria were present. One and one-half months later, the patient was neurologically negative in all respects and was discharged from the hospital shortly thereafter.

COMMENT. This case falls into the category referred to as spinal epidural granuloma. The most remarkable feature is its chronicity, covering a period of twelve years between the probable onset of the disease in January, 1923, and the patient's discharge from the hospital in March, 1935. Scarcely less remarkable is the fact that the wound site of the original laminectomy not only healed satisfactorily, but remained closed during a period of a decade without showing any tendency to break down. Whether or not the osteomyelitic process discovered at the second operation was present at the first operation is problematical. One point is certain, the long standing osteomyelitic process found at the second operation was adjacent to a chronic granulomatous inflammatory mass which had produced the partial physiological interruption of the spinal cord.

DISCUSSION

The seven cases of spinal epidural infection reported in this paper fall into two main groups, viz., those having a fairly acute inflammatory process and designated by the term "spinal epidural abscess" and those having a more chronic, low grade type of inflammatory lesion, designated as "spinal epidural granuloma." Cases I, II, III, IV and VI belong to the former group and Cases V and VII to the latter.

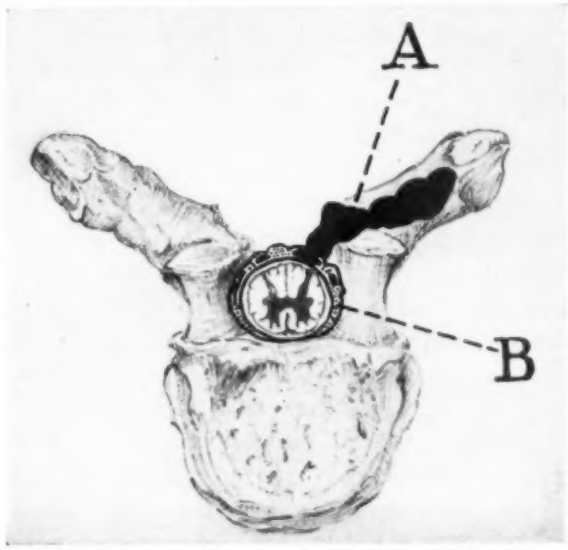


FIG. 7. Case VII, patient F. O. Diagram illustrating a destructive osteomyelitic process in the stump of the lamina of the left side of the third thoracic vertebra, A. Tough granulation tissue, relatively vascular and containing small pockets of pus, occupied the epidural space, entirely replacing the fat, B.

One is likely to encounter all grades between these two extremes represented by the most fulminating, Case III, and the most chronic, Case VII, depending upon the type and virulence of the invading organism and the resistance of the host. The pathological process of the acute forms of the disease is characterized by the presence of pus, while that of the more chronic forms is characterized by the presence of low grade, sclerosing, granulomatous inflammatory tissue. This classification corresponds to the case reports in the literature of which we have been able to find 203. The present series brings the total to 210 cases, of which 156 may be classified as acute, 2 as subacute and 52 as chronic forms of the disease. The earliest reports in the literature are those of Bergameschi, 1820,[12] Albers, 1833,[4] and Bristowe, 1855.[15] A complete bibliography has been appended to the present paper in order to bring the subject up to date. No attempt to review this literature will be

made here; for this purpose the reader is referred to the excellent summaries of Donati, 1906,[36] and of Schmalz, 1925.[115]

The Clinical Picture. The sequence of symptoms and signs in the course of a spinal epidural infection occur with sufficient regularity to warrant the formulation of a definite clinical syndrome. The entity is most clearly delineated in the acute forms of the disease, but even in the chronic forms it shines through with sufficient clearness in those cases where the patient and the physician combine their efforts to reconstruct a detailed and chronologically precise history. The history will usually be positive for a previous infection. This infection may still exist at the time of the onset of the neurological symptoms, but in most cases it will have subsided. It may have been adjacent to the spinal column or remotely removed from it. Representative examples of such previous infections are: acute upper respiratory infection, otitis media, pharyngitis, tonsillitis, bronchitis, pneumonia, abscess, furunculosis, phlebitis, cellulitis, felon, pyelitis and other infections of the urinary tract, puerperal sepsis and appendicitis. At some time during the first two or three months following such an infection, and usually within the first two weeks, the patient begins to complain of a boring pain in the spine. Most commonly this is fairly well limited to the area of one or two vertebrae. It is annoying, constant, at times throbbing, made more intense by coughing, sneezing or any movement of the spine, especially jarring movements, and is frequently associated with local tenderness over the painful vertebrae. Localized swelling may or may not be present. A marked degree of malaise is noted, there are commonly complaints of chilly sensations, headache, lassitude, generalized myalgia, feverishness and sweating. The temperature may range between 101 and 104°F. and later establishes itself as of the "septic" type, with morning remissions. The pulse rate is usually elevated to about 100 per minute and the respirations correspondingly accelerated to 24 to 28 per minute. The blood count reveals a polymorphonuclear leucocytosis of from 12,000 to 25,000. At about the time of appearance of these toxic symptoms the first neurological manifestations are noted. Involvement of the dorsal roots by the advancing infectious process produces a radicular type of pain, sharp, often lancinating, and frequently associated with a "girdle sensation." The spine and neck become rigid and flexion of the head on the chest is painful. It sometimes happens that the boring pain in the spine disappears when pus breaks into the epidural space.

Evidence of involvement of the spinal cord itself may now be added to the symptoms of root and meningeal implication. Paresthesiae or dysesthesiae of one or more extremities are the usual precursors of the more dramatic and later appearing signs. Examination at this time will frequently disclose a marked degree of hyperesthesia of the affected extremities. Depending now upon the location and the degree of acuteness of the infectious process, paralytic signs may present themselves. If the lesion is about the cauda equina, or if it involves the spinal cord and is fairly acute, a flaccid paralysis may become evident with complete areflexia in those parts of the body supplied by segments below the upper level of the involved spinal cord. If, on the other hand, a more chronic process is present the paralysis will usually be characterized by weakness, spasticity and hyperreflexia. The abdominal and cremasteric reflexes are rarely elictable. Varying degrees of abdominal distention and tympany may be encountered. The loss of tonus of the abdominal musculature and the autonomic involvement of the gastrointestinal innervation underlying these manifestations are most marked in the acute cases. In association with the onset of the paralytic signs loss of control of the sphincters frequently appears. This applies especially to the bladder. Retention appears first and is often attended by loss of the urge

to urinate. Later this retention gives way to overflow incontinence with almost constant dribbling of urine. Constipation may be bothersome, particularly in the chronic cases. With the supervention of complete paralysis, fecal incontinence adds itself to the clinical picture. Priapism may be present. The sensory status, like the motor, is dependent upon the acuteness of the process and degree of involvement of the spinal cord and nerve roots. We have already referred to the early subjective complaints of paresthesia, etc.

Examination during the early period of neurological manifestations may disclose cutaneous hyperesthesia at and below the dermatome supplied by the upper segment of the involved cord. Within a short time this hyperesthesia is replaced by varying degrees of hypesthesia and anesthesia. Deep (i.e., muscle and tendon) pain may be preserved for a considerable period following loss of cutaneous pain sense. Our experience has been that acrognosis and vibratory sense persist longer than the superficial modalities of sensation and that, of the latter, tactile sensation may be preserved in whole or part even when pain and thermal sensitivity have been completely lost. A band of hyperesthesia is sometimes found at the uppermost dermatome of disturbed sensation. Evidences of disturbed vegetative function, in all likelihood usually present, are less likely to attract attention. Differences in peripheral vasodilatation above and below the implicated levels together with palpable differences in temperature are likely to be masked by the general toxic symptoms.

Lumbar puncture in a well established case yields fairly characteristic findings. Pus may be encountered in the epidural space if frequent aspiration is carried out as the needle makes its course from the skin to the thecal sac. The spinal fluid is usually clear. It may vary from colorless to distinct xanthochromia and will be shown to contain an increase in cells of from 50 to 1000 per cu. mm., usually in the lower ranges. Both polymorphonuclear and lymphocytic cells may be thrown out into the subarachnoid space. The protein content of the spinal fluid is frequently increased to 100 or more milligrams per 100 c.c., the sugar however, remaining quite normal. Culture and smear remain negative for the presence of organisms, unless the leptomeninges have been invaded. Bilateral jugular compression may indicate the presence of complete or incomplete subarachnoid block. The colloidal gold test is not characteristic, but may at times resemble the meningitic type of curve.

It is rare to find roentgenographic evidence of bony changes of the spinal column even in the presence of a well established osteomyelitis. Therefore failure to identify such a change should not render one reluctant to make a diagnosis of spinal epidural infection if other signs point in that direction. We are in disagreement on this point, with those who, like Westerborn,[137] assert that negative radiographic films permit one to rule out vertebral osteomyelitis.

Etiology. The literature indicates that with but few exceptions, the pyogenic organism associated with spinal epidural infections is the Staphylococcus. In the Cases of Peters[98] and Schick[116] the pneumococcus was found. The pneumococcus was also encountered in Case VI of the present series. A few instances of streptococcus infections have been described. Raymond and Sicard[108] reported a case associated with the typhoid bacillus. Schmidt[117] found the Bacillus pyocyaneus and Dickson[34] and Rand[106] each reported a case of Oidium coccidioiditis. The accompanying table has been plotted on the basis of the cases in the literature and those of the present series and is intended to show the frequency with which a given portal of entry may be encountered together with the estimated time interval between the initial infection and the first appearance of either pain in the back or neurological signs. Not all the cases in the literature could be included in this table because some were merely autopsy reports, others presented

TABLE I

Mode of Infection	Author	Year	Time Interval	Organism	Character of Infection	Result
Abscess, axilla	Lowenstein (Case I)	1931	8½ weeks	Staphylococcus aureus	Acute	Died
Abscess, flank	Lowenstein (Case II)	1931	1 day after incision & drainage	Staphylococcus aureus	Acute	Died
Abscess, jaw: trauma to spine	Donati	1906	3 months	Staphylococcus aureus	Acute	Survived
Abscess, floor of mouth	Dodge	1933	3 weeks	Staphylococcus	Acute	Survived
Abscess, dental	Neale	1936	.....	Staphylococcus aureus	Acute	Survived
Abscess, periesophageal	Mixter & Smithwick (Case IV)	1932	5 weeks	Staphylococcus albus	Acute	Died
Abscess, rhomboid muscle	Putzel	1878	Few days	Thick pus	Acute	Survived
Abscess, 10th rib	Taylor & Kennedy	1923	12 days	Staphylococcus aureus	Acute	Survived
Abscess, scalp	Slaughter et al.	1934	10 days	Staphylococcus aureus	Acute	Survived
Abscess, breast following scratch by nail	Ayer & Viets	1916	3 weeks	Staphylococcus albus	Acute	Died
Abscess, thigh	Bensheim	1928	9 months	Staphylococcus	Acute	Died
Actinomycosis, jaw	Wilson & Kammer (Case I)	1934	6 months	Not reported	Acute	Died
Appendicitis, followed by subphrenic abscess and septicemia	Allen & Kahn (Case I)	1932	14 months	Staphylococcus aureus	Acute	Died
Cholecystitis followed by crop of furuncles	Schwab	1924	4 weeks	Staphylococcus aureus	Acute	Died
Carbuncle, leg	Ely (Case I)	1931	3 weeks	Staphylococcus aureus	Acute	Survived
Furuncle, arm	Veraguth & Schnyder	1929	3 years	Staphylococcus (smear)	Acute	Survived
Furuncle, forearm	Pollak	1931	12 days	Not reported	Chronic	Died
Furunculosis, chin and extremities	Morawitz (Case I)	1919	Few days	Gram positive cocci	Acute	Died
Furuncle, chin	Mintzman	1934	3 weeks	Staphylococcus aureus	Acute	Died
Furuncle, chin	Goodhart & Savitzky	1933	2 weeks	Staphylococcus aureus	Acute	Survived
Furuncle, outer ear	Fuchs	1920	4 weeks	Not reported	Subacute	Died
Furunculosis, face	Mixter	1916	2½ weeks	Staphylococcus albus	Acute	Died
Furuncle, back	Dandy (Case I)	1926	5 months	Staphylococcus aureus	Chronic	Survived
Furuncle, back	Dandy (Case II)	1926	13 months	Staphylococcus aureus	Chronic	Survived
Furuncle, neck	Allen & Kahn (Case III)	1932	Less than 3 weeks	Staphylococcus aureus	Acute	Survived
Furuncle, neck	Bunch & Madden (Case II)	1933	2 weeks	Staphylococcus	Acute	Survived
Furunculosis, general	Browder & Meyers (Case IV)	1936	7 months	Staphylococcus aureus	Acute	Survived
Furuncle, neck	Gay	1930	Several days	Staphylococcus aureus	Acute	Died
Furuncle, neck	Spiller & Wright	1921	6 weeks	Staphylococcus aureus	Acute	Died
Furunculosis, multiple, neck	Bellerose & Amyot	1932	5 weeks	Staphylococcus albus	Acute	Died
Furuncles (3) mastoid region	Ely (Case II)	1931	3 weeks	Staphylococcus aureus	Acute	Survived
Furuncle, gluteal region	Ely (Case III)	1931	3 weeks	Staphylococcus aureus	Acute	Survived
Furuncle, rectum & thorax	Hassin	1928	1–2 weeks	Staphylococcus aureus	Acute	Died
Furuncle, wrist	Schoenwerth	1902	Few days	Staphylococcus aureus	Acute	Died
Gonorrhea with orchiditis	Luce	1914	8 years	Not reported	Chronic	Died
Gonorrhea, acute rheumatism, ulcerative endocarditis	Delorme	1892	2 months	Staphylococcus aureus	Acute	Died
Blister of hand, septic carbuncle of face	Mixter & Smithwick (Case X)	1932	3 weeks	Staphylococcus aureus	Acute	Survived
Blister of knee, infected	Gasul & Jaffe (Case I)	1935	5 days	Staphylococcus aureus	Acute	Survived
Infected bullet wound of spine, followed by flaccid paraplegia.	Alajouaine & Petit-Dutaillis	1931	16 years	Sterile pus	Chronic	Survived
Infected paravertebral stab wound	Barth	1901	Immediately	Staphylococcus	Acute	Died
Infection, palmar space	Watts & Mixter	1931	8 months	Negative culture	Chronic	Survived
Infection, foot with cellulitis	Guttman & Singer	1931	6 weeks	Not reported	Subacute	Died
Infection, toe	Mixter & Smithwick (Case VI)	1932	2 weeks	Staphylococcus aureus	Acute	Survived
Infection of injury over scapula	Braun	1922	1 week	Staphylococcus	Acute	Died
Infection, internal malleolus	Schmidt	1901	1 week	B. pyocyaneus	Acute	Died
Cellulitis, nose and otitis externa	Craig & Doyle	1932	2 weeks	Staphylococcus	Acute	Survived
Cellulitis, leg followed by tibial osteomyelitis	Meslier	1894	1 year	Thick pus	Acute	Died
Infection, laceration of finger	Wei Yu Lin	1928	9 days	Staphylococcus aureus	Acute	Died
Paronychia	Riese	1898	4 days	Staphylococcus aureus	Acute	Survived
Whitlow felon	Reinhardt	1909	2 weeks	Staphylococcus	Acute	Died
Mastoiditis	Mixter & Smithwick (Case III)	1932	5 days	Staphylococcus albus	Acute	Died
Otitis media, followed by septicemia	Morawitz	1919	4 days	Staphylococcus aureus	Acute	Died
Pneumonia	Wilson & Kammer (Case II)	1934	8 days	Creamy pus	Acute	Died
Pneumonia	Nonne	1902	2½ weeks	Not reported	Acute	Survived
Pneumonia	Oehlecker (Case II)	1931	Following crisis	Staphylococcus	Chronic	Survived
Pneumonia	Navach	1933	3 days following crisis	Thick yellow pus	Acute	Died

TABLE I (*Continued*)

Mode of Infection	Author	Year	Time Interval	Organism	Character of Infection	Result
Pneumonia, followed by femoral phlebitis and furunculosis	Barker	1932	2 months	Staphylococcus aureus	Acute	Died
Phlebitis of leg during pregnancy	Oppenheim	1910	2 weeks	Staphylococcus	Acute	Died
Abortion, followed by uterine infection	Browder & Meyers (Case II)	1936	6½ weeks	Staphylococcus aureus Streptococcus non-hemolytic	Acute	Died
Retropharyngeal adenitis	Monier	1932	1 week	Not reported	Acute	Survived
"Spinal trouble" for 3 weeks at 10 years of age	Cannaday	1930	7–10 years	Not reported	Chronic	Survived
Trauma to spine	Allen & Kahn (Case II)	1932	Several days	Staphylococcus aureus	Acute	Died
Trauma to spine	Bunch & Madden (Case I)	1933	5 days	Thick white pus	Acute	Survived
Trauma to spine	Dandy (Case III)	1926	8 months	Not reported	Chronic	Survived
Trauma to spine	Hunt	1904	10 days	Staphylococcus aureus	Acute	Survived
Trauma to spine	Smitt	1929	5–6 weeks	Staphylococcus aureus	Acute	Survived
Trauma to spine	Lemoine & Lannois	1882	3 days	Pus exudate	Acute	Died
Trauma to spine	Lewitzky	1877	3 days	Thick yellow pus	Acute	Died
Trauma to spine	Simon	1855	11 days	Thick pus	Acute	Died
Trauma to spine, followed by upper respiratory infection	Westerborn	1924	2 weeks	Staphylococcus	Acute	Survived
Trauma to spine	Browder & Meyers (Case V)	1936	3 days	Not reported	Chronic	Survived
Trauma to spine	Leyden	1877	3 years	Not reported	Chronic	Died
Trauma to chest	Crouzon et al.	1932	1 year	Sterile pus	Chronic	Survived
Common cold	Delearde	1900	8 days	Staphylococcus	Acute	Died
Influenza, followed by right empyema	Birch	1934	3 days	Not reported	Acute	Died
Exposure to cold and wetness	Maguire	1888	4 days	Thick pus	Acute	Died
Acute upper respiratory infection, followed by otitis media	MacDonald	1928	10–14 days	Staphylococcus	Acute	Survived
Acute upper respiratory infection, followed by bronchitis	Schick	1909	2 months	Diplococcus lanceolatus	Acute	Died
Acute upper respiratory infection and sore throat	Gasul & Jaffe (Case III)	1935	1 week	Pus with cocci	Acute	Died
Acute upper respiratory infection, followed by abscess of heel.	Browder & Meyers (Case VII)	1936	1 week	Staphylococcus aureus	Chronic	Survived
Acute upper respiratory infection	Browder & Meyers (Case I)	1936	1 week	Staphylococcus aureus Non-hemolytic streptococcus	Acute	Died
Acute upper respiratory infection	Browder & Meyers (Case VI)	1936	3 weeks	Diplococcus lanceolatus	Acute	Died
Acute upper respiratory infection, followed by acute tonsilitis.	Browder & Meyers (Case III)	1936	4 days	Staphylococcus aureus	Acute	Died
Tonsilitis	Rosamond	1932	6 months	Staphylococcus	Acute	Survived
Septicemia (Etiology?)	Ryerson	1922	8–10 days	Staphylococcus	Chronic	Survived
Septicemia (Etiology?)	Oehlecker	1931	4 days	Staphylococcus	Acute	Survived
Septicemia (Etiology?)	Turnbull et al.	1932	9 years	Staphylococcus	Chronic	Survived
Septicemia (Etiology?)	Morawitz (Case II)	1919	3–7 days	Staphylococcus	Acute	Died

incomplete histories and still others were stated or were implied to have begun in an otherwise healthy individual presenting no history of previous infection. No attempt has been made in this paper to deal with uncomplicated cases of vertebral osteomyelitis.

Pathology. When found, the character of the osteomyelitis in the vertebra is not unlike that encountered in other bones and needs no special description here. Any part of the vertebra may be the seat of the disease. The laminae are the most commonly involved structures, but in any given case, the body, the dorsal process or one of the pedicles may be affected. When the spinal epidural space is invaded by the pathological process, any degree of involvement may be established, from an abscess limited only by the confines of the space itself, to a well circumscribed, irregular, sclerosing, granulomatous tumor mass. Between these two extremes many intermediate grades of reaction may be encountered. In certain acute or subacute cases the purulent collection in the epidural space may be negligible, the infection extending directly from the vertebra through the meninges and into the subarachnoid space and spinal cord. This is more apt to be the case when the bony infection presents itself directly against

the anterior face of the dura mater, i.e., where the dura blends with the posterior longitudinal ligament of the spine. In longed over the spinal nerve roots. The outer aspect of the dura mater may show injection and evidence of hypertrophy.

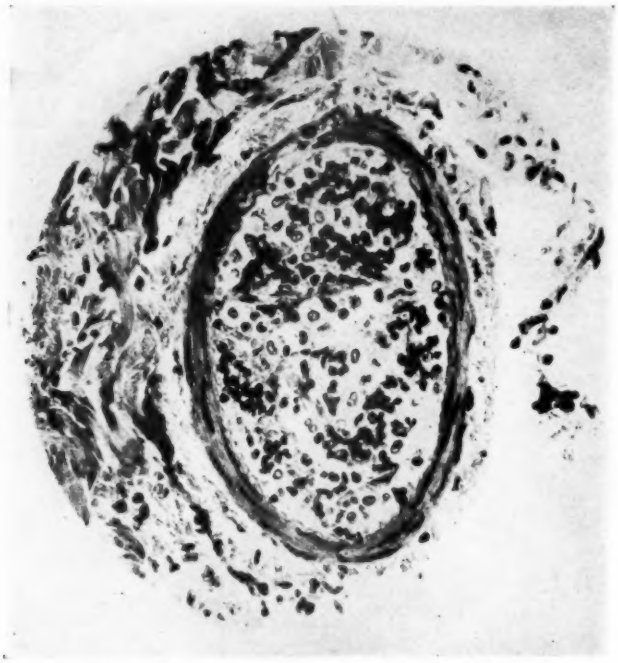


FIG. 8. Microphotograph illustrating a thrombosed vessel of the spinal cord underlying a zone of epidural abscess. A typical inflammatory and productive response with hyalinization and the outpouring of wandering cells of a meningocytic or clasmatocytic type may be seen.

other relatively acute cases a localized collection of pus may be found over one or several segments in the lateral or posterior portions of the epidural space. Extending cephalad and caudal to such a collection, the epidural fat usually undergoes reactive changes and may appear succulent, swollen, injected and perhaps necrotic. On the other hand, the productive features of a chronic case may be such as to lend the appearance of Hodgkin's disease. It is of more than passing interest that in Case VII of this series the pathologist regarded the microscopic picture as resembling Hodgkin's disease. We have noted that in several case reports (Wiersma,[139] Watts and Mixter[134] (Cases III and IV) and Chavani and David[23]), the verbal description of the pathological sections might well be applied to Hodgkin's tissue.

Those infections which are dorsally situated implicate the dura as it is pro- Wandering cells are poured out into the subdural and subarachnoid spaces, apparently marshalled for the purpose of walling off the invading bacteria. The characteristics of the spinal fluid have been noted in our description of the clinical picture. The spinal cord itself, when involved, presents a combined picture of thrombosis of vessels (Fig. 8), inflammatory response of glia cells and myelomalacia with liquefaction and vacuolization of the white substance (Fig. 9). The neurocytes themselves undergo swelling and chromatolysis and their nuclei become eccentrically displaced and may entirely disappear. Two such cells are shown in Figure 10A, and for purposes of comparison, Figure 10B illustrates normal neurocytes at an uninvolved level of the same spinal cord. Hassin[52] has very clearly described the pathological changes in the spinal cord adjacent to an epidural granuloma. Allen

and Kahn[5] have described a "honeycombed appearance of the tissue with cystic degeneration," these changes being regarded by them as the manifestations of advanced edema.

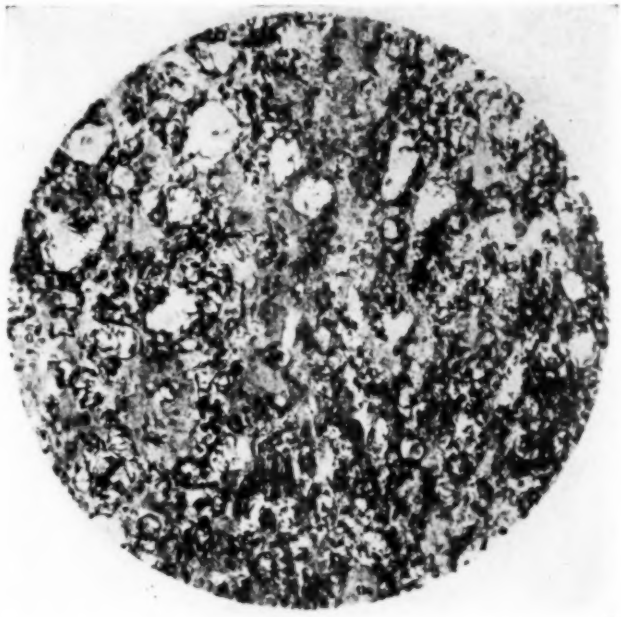


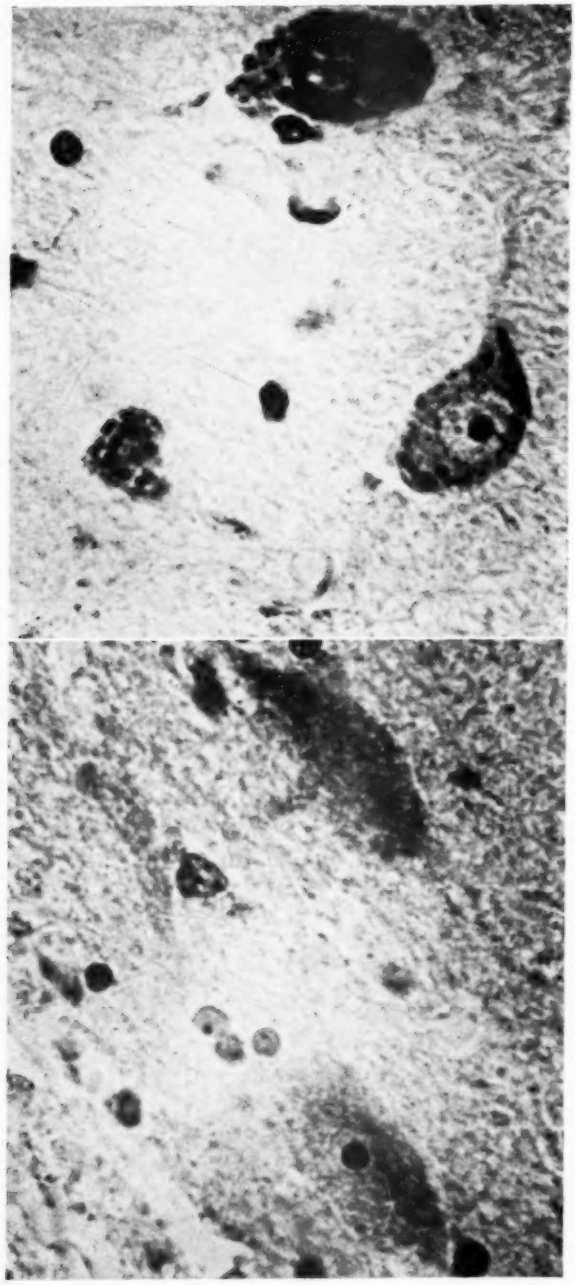
Fig. 9. Microphotograph illustrating myelomalacia in the spinal cord underlying a zone of epidural abscess. Thrombosis, liquefaction, vacuolization, myelin breakdown and gitter-cells may be seen.

Pathogenesis. According to Meslier,[80] Schmalz[115] and Dandy,[30] infection may be established in the spinal epidural space; (a) by direct extension from an adjacent infection or (b) by a hematogenous route from the lesion located elsewhere in the body. Concerning the latter mode of invasion, the great majority of writers believe that bacteria are implanted directly into the fatty tissues or blood vessels of the spinal epidural space from the blood stream (so-called "primary metastatic invasion" of the epidural tissues).

In an excellent review of the subject up to 1925 Schmalz[115] collected forty-one cases of spinal epidural infections and added two cases from his own experience. Vertebral osteomyelitis was not mentioned in connection with these cases, it being considered by this author that the metastatic infection was primary in the epidural space. This is also essentially the view expressed by Bristowe[15] who in 1855 gave the first clear summary of a case of purulent involvement of the epidural and subdural spaces. Ramsay Hunt[58] was the first to express the idea that vertebral osteomyelitis is the precursor of these epidural infections. This author in 1904 reviewed the symptomatology and pathological features of osteomyelitis of the vertebrae. Of sixty-two cases collected by him, twenty-four had epidural spinal infections associated with vertebral osteomyelitis. It seemed to Hunt that the early symptomatic features of the cases with epidural involvement corresponded so closely to the symptomatology of uncomplicated vertebral osteomyelitis that a sequential and causal relationship must exist between the two. In his concluding remarks he states, "I feel justified in asserting that there is not a single recorded case of acute primary suppurative perimeningitis which does not accord perfectly in its clinical and pathological manifestations to our present conception of acute osteomyelitis of the spine." Donati[36] and Schoenwerth[118]

are inclined to a similar view of the pathogenesis of epidural infections.

In general, however, the idea expressed by Hunt has not found wide favor. That infections of the epidural space borne by the blood stream are preceded by a zone of osteomyelitis in the vertebral column has been proved in only a small number of instances. In our own series, a definite vertebral lesion was discovered in six of seven cases. In the remaining case the data we were able to gather was not sufficient to enable us to exclude such a lesion. On the other hand, it seems clear that the small area of osteomyelitis present in those cases in which it was found might readily have been overlooked. It has been indicated that the lesion in Case 1 was discovered by accident. Again, negative morbid reports based on operative findings alone are not to be considered as conclusive evidence. Finally, even at necropsy, a thorough going,



B

A

FIG. 10. Microphotographs of neurocytes, A, from region of the spinal cord underlying a zone of epidural abscess, illustrating chromatolysis, swelling, loss of cell outline and eccentric position of the nucleus, with early pyknosis. B, from uninvolved region of the spinal cord, illustrating normal neurocytes.

detailed exploration is required to reveal a small area of bone necrosis. The bony lesion may be overlooked or subordinated to the more extensive purulent or granulomatous process surrounding it. It is probably not unusual that the examiner, having discovered an "adequate" pathological process to account for the patient's death, should fail to carry his search further, especially when this calls for a tedious chiselling out of the vertebral column. Generally, we have been impressed in our review of the literature, that a small zone of vertebral osteomyelitis has been excluded in most instances on very scanty evidence.

It is of interest to speculate on the relationship of trauma to the disease process under consideration. Schmalz[115] expressed a belief that a trauma to the back may cause bleeding or other response in the epidural space, thus creating a "locus minoris resistentiae." He asserts that years after the apparent healing of such a lesion, blood borne bacteria may find a seat of predilection here. While we cannot endorse this view, inasmuch as we are at variance with Schmalz concerning the pathogenesis of the disease, still it must be allowed that there are certain attractive features in the idea of the "locus minoris resistentiae" if this can be made to apply to the vertebral column, rather than to the epidural tissues. This is not without its parallel in the pathogenesis of osteomyelitis of the long bones. So far as the pathogenesis of cord symptoms is concerned Hassin[52] concludes that the spinal cord reactions simulate those produced by experimental compression of the neuraxis by an extradural mass. On the other hand, in only one of our series (Case VI) was there any visible evidence of cord compression. It is therefore fair to question, as have Hassin,[52] Elsberg,[39] Allen and Kahn,[5] whether there are not other factors at work than compression alone. We have observed thrombosis of the surface veins and of the spinal cord adjacent to the epidural infection. Such a pathological finding may depend upon the surrounding septic process rather than a compression factor. Although the spinal cord has a relatively rich blood supply, extensive thrombosis of its vessels in the course of a spinal epidural infection must have a distinct bearing on the physiological interruption of function so frequently noted.

Diagnosis. The most frequent error underlying the failure to diagnose spinal epidural infection is that it is not entertained as a possibility. It has seemed to us that the disease has not been given its due emphasis in textbooks and clinical considerations. Thus, in French's "Index of Differential Diagnosis," one of the standards in the English language, it is not mentioned as a cause of paraplegia. The history of the prior infection followed by severe boring pain in the back, toxic symptoms, paresis or paralysis and meningitic symptoms, should suggest the diagnosis at once. The neurologic finding of incomplete transverse myelitis is important, but by no means indispensable. Slaughter, Fremont-Smith and Munro[124] have recently demonstrated a case of spinal epidural abscess correctly diagnosed and operated upon before the appearance of neurological symptoms or signs.

A few remarks concerning the technique of lumbar puncture in these cases are in order at this point. If a spinal epidural abscess is suspected, the spinal needle should be introduced with caution. At every 0.5 cm. advance of the needle in its course through the tissues of the back, the stylet should be withdrawn and the needle aspirated, particular caution being exercised after puncture of the ligamentum flavum. If pus is encountered before the needle perforates the subarachnoid space, the diagnosis is thereby established and the needle may be withdrawn. Mixter and Smithwick[84] have emphasized the danger attached to the mechanical deposition of pus in the subarachnoid space, the point of the needle having passed through an epidural infection. If no pus is found the spinal puncture may be completed and

the Queckenstedt test performed. The findings of complete or incomplete block, xanthochromic fluid, pleocytosis, increased protein, normal sugar and absence of organisms by smear and culture should all but clinch the diagnosis. Partial or complete block will, in a majority of instances, antedate the meningitic signs and the signs of altered spinal cord function by several days.

Superficially, spinal epidural infections may be mistaken for many conditions, since, as has been shown, all ranges and degrees of involvement may be encountered. The chronic spinal epidural infections may be mistaken for other compression lesions of the spinal cord but we know of no disease that simulates a metastatic osteomyelitis of a vertebra with secondary invasion of the spinal epidural space and subsequent implication of the spinal cord.

TREATMENT

Once the diagnosis of spinal epidural infection is made, laminectomy should be carried out and as extensive an exposure made as seems necessary to permit free drainage of the infected epidural space. In the chronic form, where the epidural mass is firmly adherent to the dura, attempts at removal of this sclerosed tissue may result in the opening of the dura. This is surgically undesirable but not necessarily followed by meningitis. Probably the best method, in those instances in which the epidural mass cannot be completely removed, is to divide the mass in its longitudinal axis with the expectation of dealing to a degree with the factor of spinal cord compression.

SUMMARY AND CONCLUSIONS

1. Seven cases of spinal epidural infection have been presented. Of these, five belong to the group designated as spinal epidural abscess, representing the relatively acute process, and two belong to the group designated as spinal epidural granuloma, representing the more chronic process. Five cases died and two are living at present.

2. The clinical picture is fairly constant and, for the more acute forms at least, constitutes a fairly definite syndrome, consisting of a history of a previous infection, boring pain in the spine, radicular pain and finally spinal cord involvement. In the chronic cases there is less definition of the syndrome, although the features outlined are often represented if an adequate history is obtainable.

3. A consideration of the pathogenesis of the disease reveals two main modes by which the spinal epidural space may be invaded: in the first, by direct extension from an infection adjacent to the vertebral column: in the second, by a hematogenous route from a more remotely situated focus of infection. With respect to the latter mode, we have proposed reasons for believing that cases of this kind are characterized by a zone of metastatic vertebral osteomyelitis, which infection subsequently spreads into the spinal epidural space.

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HYPERPARATHYROIDISM

HISTORY, ETIOLOGY, AND CLINICAL PATHOLOGY

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A DECADE ago, we had small proof of the relationship between parathyroid activity and the metabolism of phosphorous and calcium, but following closely upon the heels of the isolation of the active principle of the secretion of the parathyroid glands, parathormone, and its experimental use both in animals and man, experimental pathology, histology, physiology and chemistry quickly directed the surgeon's knife toward a tumor which is usually neither visible nor palpable. This development runs parallel with exploits of surgery in the case of tumor of the islet tissue of the pancreas and the recognition of the basophilitic adenoma of the pituitary bodies. The diameter of these adenomata may be measured in a few millimeters, yet the associated constitutional disturbance frequently alters the entire aspect of the body or leads to its total destruction. The recognition of hyperparathyroidism as a clinical entity is one of the notable achievements of recent experimental medicine.

Clinical interest moving in advance of scientific investigation has led to an attack upon the parathyroid glands as a possible etiological factor in many obscure clinical conditions, especially those of the osseous system. From the results of carefully controlled clinical and experimental studies, it would appear that hyperfunction of the parathyroid glands gives rise only to a characteristic clinical syndrome consistent with the constant finding (in proved cases) of a negative balance in the metabolism of calcium. Reference to this syndrome in the literature is under the captions of hyperparathyroidism, parathyroidism, Mandl-DuBois' disease, osteitis fibrosa, osteitis fibrosa cystica, osteodystrophia cystica, osteitis fibrosa osteoplastica, osteitis fibrosa cystica generalis or diffusa, osteodystrophia fibrosa cystica generalisata of von Recklinghausen, or simply, von Recklinghausen's disease.

HISTORY

The history of classical hyperthyroidism does not carry us back to the ancient Greeks, but merely to 1926 when the relationship between the parathyroid glands, calcium metabolism and certain pathologic conditions of bone was first recognized. Previous to that date, the end results of this disease had been accurately and extensively described but its association with hyperfunction of the parathyroid glands had not been recognized.

Von Langendorff and Mommsen in 1877 and Virchow in 1886 gave very good pictures of osteitis fibrosa cystica, mentioning shortening of the patient's skeleton with softness of the bones and the occurrence of localized areas of so-called "brown cysts." Virchow especially stressed the point that these areas were not sarcomas but were areas of softening, not neoplastic in character. In the same year, 1886, Hirschberg gave a vivid description of generalized osteitis fibrosa cystica but he did not recognize it as a separate entity and classified it as a form of osteomalacia. Von Recklinghausen, five years later in 1891, in the volume commemorating Virchow's seventy-first birthday, contributed a paper entitled "Osteitis, Osteomalacia and Osteoplastic Carcinomatosis" in which appeared the first accurate description of the disease which now bears his name. This contribution to the Festschrift is very involved, but there can be no doubt that his Cases V and VII constitute the discovery of generalized osteitis fibrosa cystica. It is interesting that he carefully described 2 cases of puerperal

osteomalacia (Cases VIII and X) in order to place on record that "so many facts differentiate case seven from true osteomalacia." He described his Case VII as "osteitis fibrosa with marked bone softening and multiple osteosarcomata." The patient was a man of forty years, thought to be suffering from osteomalacia. There was a history of eighteen months of bone pain with bowing of the bones and multiple spontaneous fractures. At necropsy the bones were soft and deformed so that with a saw they cut like rotten wood and the shafts could be cut with a knife using moderate force. All the bones in the skeleton were affected and seemed histologically to be formed of tough connective tissue. Brown-red projecting tumors were found in the fibula, iliac crest, and upper and lower jaws. In his earliest description von Recklinghausen regarded the disease as an inflammatory process, a chronic productive inflammation, whereby fibrous tissue was substituted for osseous structure, resembling in this way fibrous myocarditis or hepatic cirrhosis. In his later works, however, he changed his belief and thought that he was dealing with a metaplastic condition of the existing tissue, together with calcium withdrawal.

In 1895, Mickulicz also described the condition of osteitis fibrosa cystica as a definite type of bone tumor of obscure inflammatory origin. Even as late as 1910, Bloodgood stated that the basic cause of the osteitis fibrosa was a low grade infection.

The earliest reference we have to tumors of the parathyroid glands are those of DeSanti in 1900 and Benjamins in 1902, but occurrence of pathologic changes of bones was not mentioned in either of these cases. Erdheim in 1903, Julst in 1904, and MacCallum in 1905, also each reported a case with no mention of skeletal changes.

Thirteen years after von Recklinghausen first described generalized osteitis fibrosa cystica, an important step was made towards solving the problem of its etiology. This was a description by Askanazy in 1904 of a case of generalized osteitis fibrosa associated with a tumor of one of the parathyroid glands. Since its significance was scarcely suspected, the parathyroid tumor mentioned in this case passed unnoticed. However, the descriptions of such tumors with abnormal skeletal changes by Weichselbaum in 1906, and by von Verebely in 1907, gave Erdheim a basis for his hypothesis that hypertrophy of the parathyroid glands was an inadequate attempt at compensation for an unknown function (Erdheim's hypothesis). In the same year (1907) he described 3 cases of osteomalacia associated with enlargement of the parathyroid glands. Thompson and Harris described a similar case in 1906. Seven cases of parathyroid tumors without mention of skeletal changes were collected and one of his own added by DaCosta in 1909. Bauer in 1911, reported a case of adenoma of the parathyroid gland with a moderate degree of osteomalacia. In 1913 Molineus described osteomalacia in 3 elderly females, 2 of whom had solitary parathyroid tumors, while the third had two distinct tumors. Harbitz in 1915 noted the "relationship between enlargement of the parathyroid glands, rickets, and other diseases affecting the bones." Tumors of the parathyroid gland have been reported as coincident findings in several diseases. Bergstrand in 1921 reported the occurrence of tumor of the glands in certain cases of nephritis, tetany, epilepsy, eclampsia and osteomalacia. In 50 cases of nephritis, he found one or more enlarged parathyroid glands. Also in 1921 Hubbard and Wentworth reported a case of osteitis fibrosa cystica with metastatic calcification, chronic nephritis and hyperplasia of the parathyroid glands. Other authors, as MacCallum and Dawson and Struthers, made similar observations relative to nephritis, the latter reporting cases of generalized osteitis fibrosa in which areas of metastatic calcification was found in the lungs, stomach, kidneys, and myocardium.

In 1925, Hoffheinz collected from the literature 45 cases with measurable enlargement of one or more parathyroid glands. Of these, 27 were associated with definite skeletal disease, including 17 of generalized osteitis fibrosa cystica, 8 of osteomalacia, and 2 of rickets.

To Erdheim's hypothesis that the glandular hyperplasia was an effort to prevent the excessive excretion of lime salts, Dawson and Struthers in 1923 added the suggestion that this hyperplasia was primarily a response to the development of an excess amount of guanidine, which they considered to be the primary toxic factor.

The first intimation that hyperparathyroidism might be the primary cause of generalized fibrocystic disease of bones through withdrawal of calcium appeared in 1915, when Schlagenhaufer advised parathyroidectomy in 2 cases of generalized fibrocystic disease associated with tumor. However, it was not until 1925, when Mandl of the Hochenegg Clinic of Vienna proceeded to test Erdheim's theory by transplanting parathyroid tissue into a patient with generalized fibrocystic disease. The patient's condition became worse. He then removed the transplanted parathyroid tissue and also a parathyroid tumor, and there immediately followed improvement of the fibrocystic disease. Gold in the von Eiselberger Clinic of Vienna performed a parathyroidectomy in 1927, with a similar gratifying result. Barr and his co-workers had a parallel case in 1929. Since this time there have been reported over 100 cases of generalized fibrocystic disease of bones associated with parathyroid tumors, in each of which the tumor was removed, with subsequent general improvement and recalcification of the bones.

ETIOLOGY

Before experimental and much clinical data had accumulated, Bloodgood and others proposed the theory that osteitis fibrosa cystica had an infectuous etiology. But since cultures and animal innoculations have given uniformly negative results and the pathologic picture of the disease has been produced experimentally only by methods in which infection has played no part, this theory has been discarded.

Jaffee et al. produced skeletal decalcification in dogs by feeding ammonium chloride and found that when demineralization progressed slowly and was not severe there resulted only generalized de alcification of the bones (osteoporosis), but when it progressed more rapidly, then fibrosis of the marrow (osteitis fibrosa) developed, in addition to the general decalcification.

From quantitative studies of the guanidine of the blood and urine of the patients, Koch was led to advance the theory that the symptoms resulted primarily from guanidine intoxication. By the injection of guanidine salts, he was able to produce tetany. The full significance of these observations are still undetermined.

That there is a direct relationship between the function of the parathyroid glands and the regulation of the metabolism of calcium and phosphorus has been established by an abundance of both clinical and experimental data. Hyperfunction may result from an adenoma or merely from hyperplasia of the glands but the etiology of these sources of hyperfunction is unknown. The opposite disturbance, hypofunction, resulting from ablation of a portion or all of the parathyroid bodies gave the first indication of a relationship between these glands and calcium metabolism. Thus, MacCallum and Voegtlin in 1909 demonstrated that in dogs removal of the parathyroid glands caused a marked reduction of serum calcium and symptoms of tetany when calcium fell to 50 per cent of normal. Later, it was found that these symptoms could be relieved by the administration of calcium and in 1922 it was shown by Luckhardt and Gildberg that hypercalcinemia from the intravenous administration of large quantities of calcium caused relaxation of muscles and often such

a degree of hypotonia that they hardly functioned. There also resulted calcification in various organs, especially in the kidneys and impaired renal function when calcium was given daily for several weeks.

The greatest impetus to investigation and advance in knowledge came with the isolation by Hanson in 1924 and Collip in 1925 of the active principle of the secretion of the parathyroid gland (parathormone). Since then, it has been demonstrated repeatedly that the injection of the extract causes an elevation of the serum calcium. Collip in 1925 reported an increase in the blood phosphorus in parathyroidectomized rabbits. Greenwald and Gross in 1925 and again in 1926 showed that daily parathormone injections of one hundred units in animals elevated the serum calcium and caused excretion of calcium and phosphorus in the urine, the former to as much as six times normal. Hunter and Aub in 1926, also found the same hypercalcemia with increased calcium excretion in man. Albright et al have demonstrated a negative calcium balance in animals and in man receiving parathormone injections. Finally, the disease, both gross and microscopic, of generalized fibrocystic disease of bones has been produced in animals repeatedly by Jaffe, Bodansky and Blair and also by Bryom working with Hunter and Turnbull. More recently, Jaffe (1931) and Johnson and Wilder reported that repeated injections of parathyroid extract in puppies and young rats produced uniform lesions characteristic of generalized fibrocystic disease of bone, and concluded that the disease observed in man was due to an oversupply of parathyroid hormone with consequent loss of calcium from the bones.

Thomson and Collip have reported the result of overdosage of the extract. There is marked elevation of the blood calcium, marked diminution of blood phosphorus and chlorides and death preceded by nausea, vomiting, hematemesis, diarrhea, failure of renal function and uremia.

Prolonged administration of sublethal doses produces not only characteristic lesions of the bones, but also anorexia, calcification of the kidneys and impaired renal function. Brehme and Gyorgy in 1927, found that parathormone lowered the blood phosphorus and shifted the *p*H of the blood toward the acid side without affecting the carbon dioxide combining power of the blood. Reiss and Aub in 1928 also found independently that parathormone lowered the level of the serum phosphorus. This appeared to be the first action, the elevation of the serum calcium following only after a long latent period, so that these investigators suggested the possibility that the effect on the phosphorus might be the primary one.

Studies of the influence of parathormone on the excretion of calcium and phosphorus has demonstrated more striking results than would have been expected from a comparison with its effect upon the blood. In normal individuals a constant dose of parathormone causes the phosphorus excretion to rise abruptly to a maximum in the first three day period, while the maximal increase of the calcium excretion follows more gradually. After discontinuing parathormone, the phosphorus excretion falls to normal more abruptly than does the calcium according to Albright et al. This early rapid excretion of the phosphorus may account for the primary fall in the plasma phosphorus and is further suggestive evidence of the contention of Greenwald of the primary influence of phosphorus in regard to tetany.

Although parathormone invariably raises the calcium content of the serum and urine in normal persons, there is not a proportional variation to the dosage in this response. A daily dose of fifty units of parathyroid extract is usually found to have little influence on the level of the serum calcium, yet it approximately doubles the excretion of calcium and phosphorus in the urine. When one hundred units a day are given to normal individuals the calcium level of the serum is raised

to an average of 12.5 mgm. per cent, and there is an average rise in the urinary calcium excretion to six times the normal level. The evidence available both from animals by Greenwald and Gross, and from man by Hunter and Aub, points to the conclusion that these excessive quantities of calcium salts are derived from the bones. In 1931 Johnson and Wilder not only confirmed these observations but also demonstrated that the effects of prolonged administration of parathormone are not materially influenced by simultaneous administration of irradiated ergosterol, which indicated that this disease is not related etiologically to the vitamin D deficiency disorders, such as osteomalacia and rickets.

Recently, Pomenburg and Ginsburg have treated 2 cases of thrombocytopenic purpura by inducing hypercalcemia. Both were promptly cured.

Despite all of this confirmed and apparently conclusive experimental and physiochemical data, there remains the question of the existence and identity of a primary factor which excites the parathyroid glands to overactivity. Such an eminent observer as Lang has taken a rather radical stand in refuting conclusions based upon obvious facts. As late as 1932 he stated that osteitis fibrosa was a secondary condition of the bone, dependent on circulatory disturbances following either trauma without tearing of the periosteum or functional bending and cracking of the skeleton because of insufficient calcification (rickets, scurvy or osteomalacia). "Occasional occurrence of osteitis fibrosa associated with tumors of the parathyroid gland does not contradict the concept which regards osteitis fibrosa as a secondary process in bone, dependent on osseous circulatory disturbances." In the same year Semroth and McClugage stated that "It remains an open question whether in the pathogenesis of osteitis fibrosa cystica, hyperparathyroidism is as indispensable etiologic factor as suggested by experimental results or a secondary aggravating phenomenon."

Ballin states, "It is remarkable how many of the real parathyroid cases give histories of dislike of milk, thus, with the absence of the chief 'calcium-supplier' of our diets, the question of primary dietary calcium deficiency, hypocalcemia, should not be disregarded."

Wilder has called attention to the regional distribution of cases noting that a preponderance of cases have been reported from the North Atlantic states. He suggests the possible relationship to a deficiency of vitamin D leading to hyperplasia of the gland. This contention is given added support by the fact that an unusually large number of cases have been observed in England, where there is less exposure to sunlight.

With each addition to our knowledge, the interrelationship between the endocrine glands becomes more complex. Adding to the confusion are such statements as that of Ortenberg, who in 1933 stated that "the hormone responsible for direct excretion of calcium is the thyroid." He continues to relate that nearly all of the reported cases of parathyroid tumors had an associated goitrous condition. This was not a common association in the cases reviewed by us. However, a mild degree of osteoparosis and an increase in the urinary excretion of calcium in patients with thyrotoxicosis is a rather constant finding whatever its significance may be.

The extensive literature which has accumulated in recent years on the relationship of the hypophysis to the other endocrine glands contains only meager clinical and experimental data of its relationship to the parathyroid gland. Erdheim in 1903 reported a case of acromegaly in which tumors of both the parathyroid and pituitary glands were found at autopsy. In 1912 Cushing, in a monograph on the pituitary body, described a group of cases showing primary hypophyseal disease with associated alteration in other members of the endocrine series, and in his more recent writings with respect to pituitary basophilism

describes the frequent occurrence of kyphosis, lordosis, multiple fractures, marked weakness, plyuria, decrease in height, renal changes, albuminuria and gastric pains. He states that . . . "bones soft enough to cut with a knife (osteoporosis) compression of vertebrae, and healed fractures were commonly encountered." Thus, superimposed, are the classic manifestations of hyperparathyroidism.

Also, in 1912, Schmorl reported a case of typical osteitis fibrosa cystica in which he found the association of a large basophilic adenoma of the hypophysis with diffuse adenomatous hyperplasia of the parathyroid glands. He did not suspect a relationship between the two adenomas, but Molineus, in reporting the same case in greater detail, made the suggestion of such a possible relationship. He states that "acromegaly was absent in this case because of the disease in other glands (i.e. parathyroids)." Erdheim stated that he had seen the association of osteitis fibrosa cystica and basophilic tumors of the pituitary in 2 cases at necropsy. Lloyd in 1929 reported a case with post-mortem findings of a tumor-like enlargement of the parathyroid glands and one of the islets of Langerhans in association with an hypophyseal tumor. No clinical data were available in that case from which to decide whether the parathyroid or islet tissue had had excessive functional activity during life.

Evans in 1933 in a discussion of the functions of the pituitary gland summarized the situation with regard to the parathyroids as follows:

> There seems to be no question but that we have to do with a disturbance of calcium metabolism after hypophysectomy as reflected in the decreased calcium content in the blood; whether this is due to the correlated subnormality of the parathyroid glands remains to be established. Changes in the parathyroids as a result of hypophysectomy have not been adequately studied as yet. It is difficult to view the cessation of growth of the skeletal system after hypophysectomy as essentially due to disturbance in the inorganic salt metabolism; it is more justifiable to refer it to a singular failure in cartilage proliferation in the epiphyseal discs and, of course, the dwarfism is participated in by all the organs and tissues of the body.

In 1934, Hoffman and Anselmino demonstrated in experimental dogs and rats that the calcium content of the extract from the anterior lobe of the hypophysis, is thermolabile, and in parathyroprival rats the extract did not produce an increase in the calcium content of the blood. They observed that the parathyroid glands enlarged two to three times on injection of anterior pituitary substance, with the development of a preponderance of clear cells over dark cells, also oxyphil, and a strong vascular reaction. Their conclusion was that the increase in the blood calcium was the effect of the extract of the anterior lobe of the pituitary and due to the activation of the parathyroid gland with consequent increased secretion of parathormone. Collip in 1935, reviewing the literature up to March of 1935, showed that Hertz and Kranes have obtained a fraction of beef pituitary that leads to active mitotic cell division and vacuolization of the parathyroid cells in the rabbit. Almost at the same time, Anselmino, Hoffman, and Herold published their results, showing that enlargement of the parathyroid may be produced in the rat by the administration of a pituitary extract. Recently Hertz and Albright have found that the urine of patients with hyperplasia of the parathyroids, but not of those with parathyroid adenoma, contains a substance that will produce hyperplasia in the parathyroids of normal rabbits. Thus the present concept of pituitary-parathyroid interrelationship, and this, in turn, with osteitis fibrosa cystica seems to have some basis.

PATHOLOGICAL PHYSIOLOGY

The normal anatomy of the parathyroid glands with some of its variances will be

discussed here, only to say that a variance in number (from two to twelve) and position (from the pharynx into the mediastinum) as reported by Churchill and Cope, is common knowledge.

The histologic characteristics of these glands were first described by Welsh in 1898. He divided the cells of the normal gland into oxyphil cells and principal cells and subdivided the latter into four types according to their form and arrangement. Erdheim described two kinds of oxyphil cells, the large pale and the small dark. The oxyphil cells occur first in the tenth year, and are present in variable number thereafter. Hunter and Turnbull agree, but only distinguish the three types—the dark oxyphil, the pale oxyphil, and the principal cells. The appearance suggests that the oxyphil cells are principal cells in which the cytoplasm has been overcharged with oxyphil granules, that the basophil net has been more or less completely reduced to a limiting membrane. Bergstrand in 1934 expressed the opinion that the cells in parathyroid tissue were only of one kind and that the cells of Welsh are degenerating forms in the aging.

According to Hitzrot and Comroe, clinical hyperparathyroidism may be divided histologically into two types; the primary with benign, or rarely malignant tumors of the parathyroid gland, and the secondary with functional hyperactivity and, at times, hyperplasia of the glands. In the former case, the high serum calcium, negative calcium balance, and decalcification of bone is related to the increase in the number of active parathyroid cells, while in the latter type, the etiological relationship is not so clear. It has been suggested that hyperactivity may follow a prolonged low intake of calcium, so that the glands, to keep the concentration of calcium in the blood normal, progressively remove calcium from the storehouses in the bones. Theoretically, the depletion of the bones may progress, as a result of this overcompensation long after the actual need for the stored calcium has passed. Barr and Bulger and many others have observed hyperplasia of the parathyroid gland and clinical evidence of hyperparathyroidism in many cases of generalized diseases of bone, including rickets, puerperal osteomalacia, multiple myeloma and carcinomatous metastases to bones. In such instances, the hyperplasia appears to be secondary to the changes in bone. In keeping with the general provision of nature, the functional integrity of the glands of internal secretion is protected by a wide margin of safety; therefore a large portion of most glands can be removed without measurable effect on the body, also that hypertrophy of the remnants occurs if the hyperfunction demands it. Conversely, that tumors may occur without causing recognizable symptoms is reported by Lloyd who, in a recent review of 10,000 consecutive autopsies, found 5 parathyroid tumors, without histories of recognizable clinical symptoms.

As has been discussed in foregoing sections, McCallum and Boegtlin in 1909 clearly demonstrated that postoperative tetany was the result of destruction or removal of the parathyroid glandules in thyroid operations. Since they caused convulsions of tetany to cease by intravenous injection of calcium salts, they concluded that the function of the glands is to regulate the calcium exchange of the body and considered the symptoms following parathyroidectomy due solely to calcium deficiency. Calcium starvation and the resultant hyperplasia of the parathyroids can be produced by diets grossly deficient in calcium or in conditions in which there is a failure of the ingested calcium to be absorbed from the bowel. This has been demonstrated clinically in cases of chronic steatorrhea and in deficiency diseases, such as osteomalacia and rickets; also in animals with experimentally produced rickets (Pappenheimer and Munro). Some of the parathyroid glands in rachetic animals became ten times as large as normal and the micro-

scopic sections revealed benign hyperplasia similar to that described by Ballin and Morse in some of their cases.

This has given rise among the pathologists to a difference of opinion as to whether the parathyroid tumors are primary or secondary in relation to the skeletal changes of osteitis fibrosa cystica. Erdheim in 1911 defended the view that the disease of the skeleton was the primary factor and that skeletal decalcification created such a demand for calcium that a compensatory hypertrophy of the parathyroid glands occurred. Anatomical evidence against this view is found in the fact that, in generalized osteitis fibrosa, hypertrophy or tumor formation is usually confined to one parathyroid gland, the others remaining apparently normal. As a result of Erdheim's investigations the whole question was repeatedly discussed at scientific meetings in Vienna. Questions were raised at that time whether it was not justifiable to attempt the extirpation of such a tumor of the parathyroid gland in cases of generalized osteitis fibrosa, but it was not answered until Mandl in 1926, first performed this operation.

Let us now revert for a moment to the relationship between the three agents active in calcium metabolism. Dragstedt in 1927, deduced from available data that the parathyroids serve "in the transfer of calcium from the alimentary tract and its deposition in osteoid tissue during growth and repair." This is also the precise theoretical function of vitamin D. When these vitamins are supplied in great excess of the calcium intake the additional quantity of calcium (required to fulfill the functional role of the vitamins) is withdrawn from the labile stores in the trabeculae of the bone. Similar demineralization is also the result of excessive doses of parathormone. Thus the parathyroids act as the key-control in calcium metabolism subject to action of vitamin D, and it has been suggested, therefore, that these glands may also serve as a compensating mechanism when there is lack of vitamin D (and, post hoc, inadequate absorption of exogenous calcium). This compensatory overactivity of the glands for the release of calcium from the bones for use elsewhere may result in chronic hyperfunction (hyperparathyroidism). Ortenberg believes that the normal function of the parathyroids may be a positive anabolic factor in calcium metabolism. He states that the hormone responsible for direct excretion of calcium is the thyroid gland, but offers no proof. It is interesting, however, in the light of his contention that callus formation, notwithstanding a calcium-phosphorous product near the optimum (above 30), is delayed by removal of the parathyroid glands. It is worthy of note that, in the generalized decalcification of the skeleton, the teeth do not take a part. They may fall out because of osteoporosis of the jaws, but they themselves remain well calcified. This is brought out distinctly by roentgenograms in which the well calcified teeth stand out sharply against the poorly calcified jaws, and according to Albright, Aub and Bauer, would seem to indicate that they do not act as a reserve supply of calcium.

The characteristic blood chemistry findings (hypercalcemia, hypophosphatemia, and the phosphatase readings) will be discussed later. However, it should be mentioned here that the occasional secondary anemia and leukopenia which develop are probably the result of the replacement of marrow with fibrous tissue, leading to a decrease in the hematopoietic function.

There is no other entity among the lesions of bone in which there has existed such variation and confusion in pathologic descriptions and especially in interpretation. Conceptions regarding their nature have ranged from that of simple repair or frank infection on one side to true neoplasm and outspoken malignancy on the other. In spite of the apparent differences regarding etiology there has been a growing tendency to consider both the giant cell tumors and osteitis fibrosa as pathologically

related, because of the number of instances of cases in which both exist simultaneously.

To the surgical generation which immediately succeeded Paget (1853) and Nelaton (1860) the myeloid sarcoma or giant cell tumor of bone appeared securely established as a clinical entity. It was sufficient merely to emphasize the distinctions between it and the true sarcoma of bone. Until the advent of von Recklinghausen's classical description of generalized osteitis fibrosa in 1891, this conception remained unchallenged. But with the ultimate widespread recognition in more recent years, that typical osteoclastomata can almost invariably be found in the varied histological pattern which characterizes the osteitis fibrosa group of lesions, the separate identity of the giant cell tumor is now by no means so obvious. For here is the seeming paradox of a histological picture shared by two lesions generically opposed—on the one hand a true tumor of bone (an osteoclastoma), which behaves in every particular as a neoplasm, and on the other hand, a dystrophy of bone, admittedly non-neoplastic, which exhibits a striking tendency toward spontaneous healing, as reported by Pratt.

Pick, in his Harvey Lctures of 1931, discussed the malacic diseases of bone as follows.

> Osteitis fibrosa cystica is called "metapoetic" disease associated with a complete alteration of structure, and the term ostitis should be replaced by osteodystrophia because no inflammatory signs are present. The pathological findings of the incipient stages of von Recklinghausen's disease are known. They appear as instances of so-called "progressive bone atrophy," a disease described by Askanazy. Macroscopically and by *x*-ray they exhibit a picture of simple osteoporosis in an otherwise quite intact skeletal system, however, microscopically the connective tissue from the endosteum gives rise to a "dissecting resorption" of the bony trabeculae.

Maximow states that it is almost certain that osteoclasts produce a lytic ferment, for, where they come in contact with the surfaces of the bony lamellae, the cells of the latter dissolve. Because the specific nature of these brown tumors and cysts was assumed a priori, they were considered as undeveloped or abortive forms of von Recklinghausen's disease. The literature is full of instances of "localized von Recklinghausen's disease." However, recent investigations indicate that they are a type of granuloma (abundance of spindle cells and excessive amount of hemosiderin with abundance of giant cells in groups), and not "giant cell sarcomata" but rather reaction-and-resorption tumors. It is believed by many that they arise upon a basis of primary hemorrhage or necrosis and that they are so frequently seen in von Recklinghausen's disease because trauma and hemorrhage occur more readily owing to the increased softness and poor mechanical resistance of the bone.

According to von Recklinghausen an extremely intensified process of bone development and destruction takes place in a marrow which has previously become fibrous. This fibrosis probably represents a proliferation of the reticular cells of the bone marrow. It is probable that the destruction occurs through the activity of mono- or multinucleated osteoclasts. Growth continues, at least at first, without the participation of the osteoblasts, by an indirect metaplasia, that is, by the conversion of the proliferated connective tissue cells of the endosteum and marrow. Because of the total irregularity in which the collagen fibrils develop about these newly formed bone cells, a totally irregular bone results. This is in marked contrast to the regular lamellation which characterizes the bone of normal adults. It appears that osteoclastic activity builds a pathway along which the invading connective tissue enters the solid bony trabeculae and there proliferates, excavating and finally replacing these trabeculae of the spongy bone. All the old bone structure disappears and the fatty and lymphoid marrow becomes fibrotic. The former compact or spongy bone is replaced by a

totally irregularly constituted bony structure. No sooner is this type of bone produced than it disappears, only to reappear again. This indisputable coincidence of the basic histologic changes in Paget's disease as well as von Recklinghausen's disease has become the source of a fundamental misconception among many pathologists.

The two prominent clinical features which characterize late von Recklinghausen's disease are the marked softening of the bones and the associated bone changes with the so-called "brown tumors" and cysts. Often there results infractions and spontaneous fracture, which in advanced cases lead to astonishing snake-like warping of the bones. In general the pathologic anatomic features of the disease are dominated by hypostosis, that is, by marked preponderance of the bone resorption. The small amount remains essentially as osteoid tissue. Here, too, however, as in other diseases characterized by resorption of bone, calcification is by no means entirely absent. Mosaic structures, when they do occur, are of orderly arrangement. Two additional secondary changes may occur, clefts may develop in the fibrous areas and enlarge until they form multiple cysts with fibrous walls. Secondly, the osteoclasts in certain areas may proliferate to such a degree that they form osteoclastomata (benign tumors of bone with giant cells), as described by Pick.

Elmslie in 1934 summarized the situation as follows:

> The name "osteitis fibrosa" has been applied to a miscellaneous group of cases of bone disease, in which the skeleton shows some or all of a certain group of pathological changes. These changes are:
>
> 1. Lacunar absorption of bone by osteoclasts either locally in one place or in several localities, or generally throughout the skeleton;
>
> 2. Deposit of new bone of an irregular texture (woven bone);
>
> 3. Fibrosis of the marrow, which, again may be local or general, and may simply mean that the marrow contains more fibrous tissue than normal or may mean that it is entirely replaced by masses of fibrous tissue;
>
> 4. The formation of cysts. These may lie in the bone with only a bony wall, or there may be a fibrous lining, or the cysts may lie in the masses of fibrous tissue.
>
> 5. The formation of osteoclastomata, i.e., giant-celled tumors, such as were formerly called myeloid sarcomata. They may be single or multiple.
>
> 6. Sometimes, the development of cartilage.

We must recognize that these are changes which may occur in the bone from varying causes; none of them is peculiar to any one disease. It is unfortunate, therefore, to find pathologists reporting that a specimen of bone shows "the changes of osteitis fibrosa cystica." Even if the clinical and pathological picture leads him to arrive at this diagnosis, he should give a detailed report upon the changes shown in every case.

Since Virchow's first description of a bone cyst in 1876 and von Recklinghausen's description of the generalized disease in 1891, an extensive literature has accumulated and a whole medley of cases of different varieties has been recorded, so that it is by no means simple to assort the cases. Nevertheless it is possible to make a series of clinical or clinicopathological pictures classing the cases in groups and giving them definite names, as follows:

1. Bone cysts, formed by lacunar absorption of bone by osteoclasts;

2. Osteoclastomata, classical myeloid sarcomata; related to cysts and occur in cases of hyperparathyroidism;

3. Osteoclastomatous cysts, soft tissue about the margin of cysts containing giant cells;

4. Diffuse fibrosis of bone, replacement of bone and marrow by massive formation of fibrous tissue in which small fragments of bone are embedded;

5. Generalized diffuse fibrosis of bone, as in 4, but occurring in many bones; can be distinguished from cases of hyperparathyroidism by clinical history and radiological findings, as well as by the

absence of general porosis of bones and the characteristic skull changes and biochemical changes found in the latter condition;

6. Hyperparathyroidism, *generalized* porosis of the entire skeleton with cyst formation and osteoclastomata, and thickening and porosity of the skull, accompanied by hypotonicity and a negative calcium balance.

It should be noted as Jaffee in 1933 pointed out, that the portions of the skeleton most susceptible to decalcification are those in which formation of bone is most active, i.e., the spongy bone of the metaphysis, especially of the long tubular bones. For this reason, decalcification during the adolescent period would be expected to give the atypical bony changes which have been reported in this group.

Calcium deposits are commonly found in many organs and tissues of the body. The most common site is the kidney and less commonly the stomach, spleen, liver, and blood vessels. Recently, calcification of the placenta has been reported in 2 cases.

SUMMARY

For several decades, preceding the present, pathologists had observed the occasional occurrence of a tumor of a parathyroid gland in patients with generalized changes in the skeleton, characterized by dimineralization, fibrocystic disease and giant cell tumors, but considered the association merely coincidental. The relationship of these osseous changes to the tumors did not become evident until the relation of parathyroid function to calcium metabolism became known.

There then followed an attempt to attribute various types of disease involving the skeleton such as osteitis deformens or Paget's disease, arthritis deformens, fragilitas ossea and localized fibrocystic disease to hyperfunction of the parathyroid glands. From both clinical and experimental data it has become recognized that hyperparathyroidism is a definite clinical entity characterized by a demonstrable disturbance of calcium metabolism. There is a generalized withdrawal of calcium from the skeleton which in many instances results in certain secondary changes designated as fibrocystic disease and giant cell tumors. This abnormal withdrawal results in hypercalcinemia, hypercalcinuria, hyperphosphouria and hypophosphonemia. Calcium outgo exceeds calcium intake referred to as a negative imbalance of calcium metabolism. Only in the presence of such evidences of a negative imbalance are we justified in making a diagnosis of hyperparathyroidism.

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ACUTE APPENDICITIS*

A COMPARATIVE SURVEY WITH REMARKS ON ITS MANAGEMENT

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ACUTE appendicitis today appears to be no less a problem than it was forty years ago. It is amazing, but none the less true, that its surgical treatment still entails a mortality which, according to our most reliable sources, actually exceeds the figure which was claimed for its medical management forty years ago. It is natural that the morbidity of the disease should increase, paralleling the increase in the country's population, but it is also a fact that the mortality has been steadily increasing. Census bureau figures prove that the mortality rate from appendicitis has increased progressively during the past thirty years, and that only during the recent few years has it shown even a slight tendency to recede. A grim touch of irony is contained in the fact that while such one-time scourges as diphtheria, smallpox and tuberculosis have shown a response to public health control that is truly remarkable, appendicitis, which in a great many sections apparently has not attained the importance of a public health problem, has continued steadily on its way, and now exacts a toll of between 20,000 and 25,000 lives annually in the United States. When we add to this requiem the tale of the million and more of people, who are disabled through its ravages for varying periods of time annually, we gain a clearer concept concerning the magnitude of the economic importance of appendicitis today.

Vital statistics form the basis for the contention that the death rate from appendicitis is on the increase. Census bureau figures for the registration states of the United States showed an increase in the appendicitis deaths per 100,000 from 10.8 in 1910 to 15.4 in 1930. Moreover, increasing death rates are quoted by life insurance companies in broad surveys of their insured, and by public health departments in various localities. The Metropolitan Life has recently reported an increased death rate among its white males of 20 per cent within nineteen years, and of 14 per cent for the same period among its white females.

In late years, there has been considerable improvement in vital statistics due to a more accurate reporting of causes of death. Better diagnosis has been made possible in the complications of appendicitis by *x*-ray fluoroscopic examinations, and various laboratory tests, with the result that many cases are now reported as dying from appendiceal complications, which doubtless would not have been suspected years ago.

The consensus of opinion is that the death rate from appendicitis is increasing. This is probably a true assumption, but from a study of the literature, one might easily be led to suspect that it is actually decreasing, judging from the many reported series showing such admirably low mortality rates. It is more than likely that most of the series having average or high mortality rates never find their way into print, chiefly because they contain nothing spectacular, and there is little incentive for their publication.

It is impossible for the average hospital, with its diversified staff to attain a mortality rate in the surgical treatment of appendicitis, which is comparable with that claimed by certain clinics or individuals. There a perfection of technique and a soundness of surgical judgment probably exist which are beyond the reach of a

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sizeable group of independent individuals, usually possessing varied surgical abilities, such as comprise the average hospital staff. As might be expected, the contrast will be most apparent in the handling of the all important group of peritonitic cases, where surgical opinions are so prone to differ, at such frightful cost of human life.

The series here presented comes from a hospital of four hundred beds, where surgery is performed by an attending surgical staff of eighteen men, as well as by a group of visiting surgeons, whose number is almost as large. The majority of these surgeons are in the younger age group. Their experience, technique, and surgical judgment vary considerably. Patients are admitted to the hospital from the city of 80,000 inhabitants, and from a manufacturing and farming district for a radius of twenty miles or more. Because many of the cases admitted come a considerable distance, and frequently are hospitalized late in the course of their illness, having either self-treated themselves beyond the time of safe treatment, or having been erroneously diagnosed by their local doctors, and because there has been no attempt at specialization of treatment, such as occurs in a few hospitals and clinics, where the handling of appendicitis is delegated to one man, or small group of men, but rather it is here the problem of a large and diverse staff; it is thought that the type of case handled at this institution and its general management is fairly representative of that encountered in the hospitals of the rank and file of localities throughout the country. Therefore, it is felt that conclusions, which are based upon experiences here, being representative, should be of value in determining the true trend in the surgical management of appendicitis today.

THE SERIES

This paper presents a series of cases operated for appendicitis at the Binghamton City Hospital during the year September, 1934 to September, 1935, and for the purpose of comparison, during the same months five years previously, that is from September, 1929, to September, 1930. Cases in which the appendix was removed routinely, or in which appendicitis did not appear to be the principal pathology are not included. Cases of chronic appendicitis and so-called subacute appendicitis are grouped under the one classification "Chronic Appendicitis." They are not important in this analysis except for their number and chance mortality. The problem of appendicitis does not lie in this group.

During these two years 777 cases were operated primarily for appendicitis. There were 20 deaths, a mortality of 2.6 per cent. Of the 777 cases, 346 were operated during the year 1929–1930, while 431 were operated during the year 1934–1935. There were 413 chronic cases and 364 acute cases. Among the 413 chronic cases, there was one death from postoperative pneumonia, which gives a mortality for that group of 0.2 per cent.

The 364 cases of acute appendicitis were selected after careful personal study of the histories and physical examinations, when available, the temperature and pulse charts, laboratory data, operative sheets and pathological diagnoses. Only those cases were considered acute where the diagnosis was certain beyond any reasonable doubt. In a report of this kind there is opportunity for difference of opinion, but the individual coefficient is probably reduced to a minimum when one man reviews all cases. The error, such as it may be, is therefore a constant one.

There were cases in this series in which the operative sheet gave a diagnosis of acute appendicitis, but where the preoperative data and pathological diagnosis did not concur. In several of those cases the diagnosis was thought more properly to be subacute or chronic appendicitis, and the cases were classified as such. In a certain few, the preoperative data, operative reports, and clinical course favored a diagnosis of acute appendicitis, but

the pathological report was subacute or chronic. In those cases the diagnosis of acute appendicitis was upheld and the pathological report considered unrepresentative. In general, however, great importance was placed in the pathological diagnosis, and it is felt, rightly so.

For the purpose of clarification, the acute cases were grouped, (1) as uncomplicated, depending upon whether the infection at the time of operation was confined to the appendix, or (2) whether it had passed through the appendiceal coats with involvement of the peritoneal cavity. It is this latter group which is responsible for roughly 99 per cent of the deaths in reported series of acute cases.

Next, they were classified according to the pathology found at the time of operation. An acceptable classification follows:

Acute Uncomplicated Appendicitis. Here the process, which may vary from the early acute catarrhal inflammation to a gangrenous appendicitis, is entirely limited to the appendix. The appendix may show congestion of any of its coats, edema, invasion by polymorphonuclear leucocytes, and there may be a clear or slightly turbid peritoneal fluid present in the abdominal cavity, which is sterile on culture.

Table I

	Cases Operated	Died	Mortality, Per Cent
Acute uncomplicated...	248	1	.4
Acute local peritonitis...	67	5	7.5
Acute with abscess.....	32	4	12.5
Acute diffuse peritonitis	17	9	52.9
Total.....	364	19	5.2

Acute Appendicitis with Local Peritonitis. The chief findings here are peritoneal inflammation in the region of the appendix. The cecum and terminal ileum present distention, injection of their serosa, fibrinous deposits, and sometimes omental adhesions. The peritoneal fluid may be slightly turbid, or frankly purulent.

Acute Appendicitis with Local Abscess. Here the abscess forms about the appendix, varying greatly in its fluid content and size. The walls are formed by the abdominal wall, mesentery, bowel, or omentum. The pus is ordinarily very foul and highly infectious.

Acute Appendicitis with Diffuse Peritonitis. This classification is not always easy to make. Strictly, it is the status of the abdomens in neglected or overwhelming cases seen at autopsy, where the inflammatory reaction is present in all, or practically all portions of the peritoneal cavity. The coils of intestine lie distended, dull, edematous, adherent by fibrinous plastic exudate, the intervening hollows and the natural gutters forming pools of pus. Practically, however, there are many cases which show gradations between the so-called local peritonitis and this generalized state, cases designated often as "spreading peritonitis," where the infectious process is advancing and will either continue to a generalized peritonitis, or subside to a localized process. At operation, it is sometimes difficult to state positively the true extent of the peritonitis unless the operator should explore and disturb abdominal quadrants, which are much better left untouched. However, by careful and gentle inspection, one can usually make a fairly accurate estimate of the extent of the infectious process.

Table II

	Cases Operated	Died	Mortality, Per Cent
1929–1930.....	176	11	6.2
1934–1935.....	188	8	4.2
Total.....	364	19	5.2

In our series of 364 cases of acute appendicitis 248 uncomplicated cases showed but one death, a mortality of 0.4 per cent. Certainly no more eloquent testimony to the safety and wisdom of

early operation in acute appendicitis could be offered. The one death, which occurred within twenty-four hours after operation showed high temperature, very rapid pulse and convulsions. The exact cause of death was not determined. In the complicated group 67 cases of localized peritonitis showed 5 deaths, a mortality of 7.5 per cent; 32 cases, with local abscess gave 4 deaths, a rather high figure of 12.5 per cent mortality; and 17 acute diffuse peritonitis cases gave 9 deaths, a mortality of 52.9 per cent. Therefore, while the uncomplicated cases showed a mortality of 0.4 per cent, the complicated group of 116 cases, with 18 deaths, gave a mortality of 15.5 per cent. The mortality for the entire series of 364 acute cases was 5.2 per cent.

AGE INCIDENCE AND MORTALITY

Acute appendicitis is primarily a disease of youth and young adulthood. The truth of this statement is clearly demonstrated by Krech's series in which 56 per cent of the cases were attacked during the second and third decades of life. In the present series 63 per cent of the cases occurred during the same two decades.

Table III
AGE INCIDENCE AND MORTALITY

Years	Total	Died	Mortality, Per Cent
1–10	46	3	6.5
11–20	128	3	2.3
21–30	102	4	3.9
31–40	42	2	4.8
41–50	15	4	26.6
51–60	18	3	16.6
Over 60	13	1	7.7

It is not from this particular age group, however, that appendicitis takes its heaviest toll of life. Appendicitis proves most dangerous to the person beyond the age of forty years. The mortality peak in our series occurred in the forty-one to fifty year group, where 15 cases showed 4 deaths, a mortality of 26.6 per cent As a rule, the oldest group, i.e., over sixty years, shows the highest mortality, with the sixth and fifth decades following. Then comes the first decade in life, followed in varying order by the second, third and fourth. This finding is consistent with the clinical experience that death in appendicitis very often is due to a profound intoxication of the cardiovascular system and the various organs. This toxemia ordinarily is best combatted by the strength and resistance found in youth and young adulthood. The youngest patient in this series was two and one-half years and the oldest seventy-four years.

SEX INCIDENCE AND MORTALITY

It has been a rather constant finding in the various reported series that acute appendicitis attacks the male much more frequently than the female. Krech, in his recent survey of appendicitis in New York City found a proportion of 68 males to 32 females. In our series, the males and females were in almost equal numbers, 186 males as compared with 178 females. More important than the sex morbidity, however, is the sex mortality. The male mortality in our series was 5.9 per cent as compared to the mortality of 4.5 per cent among the females. This difference of 1.4 per cent actually represents a 23 per cent greater mortality for males than for females. It is interesting to note that Metropolitan Life statistics for the period 1911–1915 showed a death rate from appendicitis for males which exceeded that for females by 26 per cent, and for the period 1925–1929 by 33 per cent. The cause and significance of this sex

Table IV
SEX INCIDENCE AND MORTALITY

	No.	Died	Mortality, Per Cent
Males	186	11	5.9
Females	178	8	4.5

variation in mortality are not clearly understood.

SEASONAL VARIATION

There appears to be considerable variation in the seasonal incidence of acute

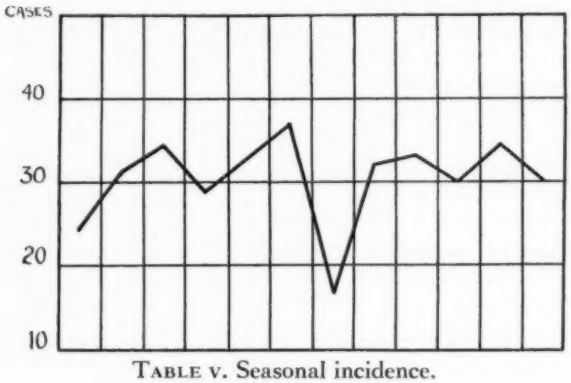

TABLE v. Seasonal incidence.

appendicitis. It has been the teaching that the incidence of appendicitis rises sharply during the late summer months, when the various fruits are ripening. The seeds and other indigestible portions of those fruits are considered important etiological agents because of the mechanical irritation which they may produce if they lodge within the appendix. It appears to be a scientific fact that the incidence as well as the mortality of acute appendicitis has a definite relationship to the occurrence of the common cold and other respiratory infections. The graph representing case incidence, according to months in this series was fairly evenly sustained during most of the months of the year, but showed a sharp decline for the midsummer month of July. This coincides roughly with the observation of Ramsdell, who, in a recent series, noticed a definite drop for June, July and August, with August being the lowest level reached. Unquestionably, there is considerable seasonal variation, both in incidence and mortality.

PREVIOUS ATTACKS

When histories of appendicitis cases are accurately taken, it is possible to compare the number and percentage of "first attack" cases occurring during successive years of a series, and to draw reasonable conclusions regarding the appendix-consciousness of the general public. For example, if the percentage of patients experiencing their first attack during a given year, shows a sharp increase over the number for a previous year, it is fairly reasonable to assume that the campaign, being waged for earlier medical attention of abdominal pain is achieving results. Unfortunately, failure to consider previous attacks in the records of a considerable percentage of the cases in this series has made any such study worthless.

CATHARTICS

Bower and others in Philadelphia investigated thoroughly the subject of cathartics in acute appendicitis. The very creditable reduction in mortality, which followed, among other things, their anti-cathartic campaign is eloquent proof of the improvement that is possible through progress in that aspect of the problem alone. In spite of the fact that for years teachers in medical schools and hospitals have emphasized the importance of the connection between "purgation, perforation and peritonitis" in appendicitis, the case records in this series were so completely lacking in positive information on this subject as to make any worthwhile conclusions impossible. However, a considerable proportion of the fatal cases had a history of cathartics, and the general impression gained in this survey is that cathartics and enemata may have played a very important part in the mortality of those cases.

INCISION AND DRAINAGE

It was hoped that some worthwhile conclusions could be reached in this series regarding the best type of incision, and the use of drainage in acute cases, especially since each of these subjects is the center of considerable discussion and controversy today. The verbal battle waxes strong

between the proponents of the McBurney as opposed to the right rectus incision, and it may be said that the evidence strongly favors the use of the gridiron incision routinely as a measure of considerable importance in decreasing operative mortality. Reid reported a startling reduction of almost 5 per cent in the operative mortality of appendicitis at the Cincinnati General Hospital since the inauguration of the routine McBurney incision. In his two reported series, the duration of illness and the incidence of purgation were roughly the same, so that the control is quite satisfactory. However, no credit is claimed for the surgical judgment and skill of the group whose efforts produced that excellent mortality drop, a point which seems worthy of consideration.

Many surgeons are advocating and practicing tight closure of the abdomen in cases where local and even early diffuse peritonitis is found. Cottis and Ingham report not only a much smoother and shorter convalescence in non-drained cases of this type, but they present mortality statistics which compare very favorably with the best offered in the more satisfactory drained series.

Shipley states that ileus and mechanical intestinal obstruction occur much more frequently following the use of abdominal drains, and he concludes from his experience that early peritonitis can be treated by tight abdominal closure, without increase in its death rate.

Only recently Gamble reported his "wide open" treatment of ruptured appendicitis by which he has obtained a mortality rate in severe peritonitic cases that is truly amazing.

Coffey uses the rather complicated quarantine drain in severe appendicitis cases, and Kennedy reports good results from his radical peritoneal toilette followed by the insertion of a bulky coffer dam drain of gauze.

Dorrance and Nealon have been using cecal drainage in selected serious cases of ruptured appendicitis, and they now report that they are inclined to use it in all ruptured cases where drainage is indicated.

Thus it is apparent, that worthwhile statistics on these subjects should be extremely interesting and instructive. Unfortunately, the operative reports in this series were so incomplete that in less than half of the cases was the type of incision given. The figures for drainage are more informative. Of the 364 acute cases 154 were drained. Drainage was performed in 111 of 116 complicated cases as well as in 43 uncomplicated cases. It is apparent that in 5 cases of peritonitis, it was thought that the peritoneum was capable of handling the infection without abdominal drainage. The use of drainage in 43 uncomplicated cases as in accordance with the views of the more conservative teachers who have taken the stand that it is better to be safe than sorry in the matter of drainage. Naturally, this number includes gangrenous and suppurative cases, many of them, no doubt, with serous peritoneal exudation. These are the cases in which, according to many surgeons, tight closure of the abdomen is not only quite safe, but is followed by a postoperative convalescence which is smoother and less protracted than when drainage is used.

TIME ELEMENT

It has been established by investigators of this subject that the chances of a fatal outcome in this disease increase in direct proportion to the time elapsing between the onset of symptoms and operation. In other words, the patient's chance of recovery depends almost entirely upon the speed with which he comes to operation. In Krech's recent study, the rise in mortality was very sharp, beginning with the group having a history of from forty-eight to seventy-two hours duration.

In 130 cases of this series where specific data on the time interval were obtained, 91 cases admitted during the first forty-eight hours of their illness showed only one death. In sharp contrast to this low

mortality in the early stage, are the 4 deaths (7 in the series), which occurred in the group of 14 cases having histories

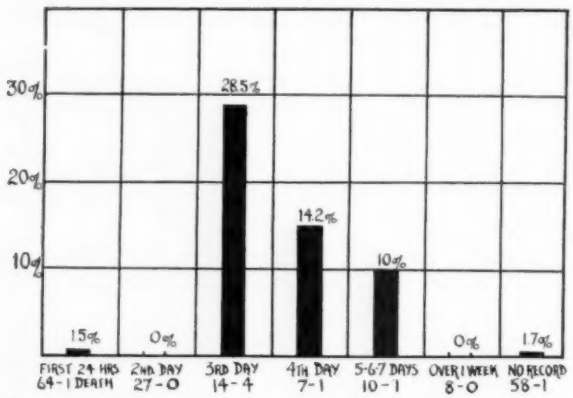

TABLE VI. Mortality in various time groups based on time elapsed between onset and admission, 1934–1935.

of forty-eight to seventy-two hours duration. In other words, 10 per cent of the cases in this series accounted for over 50 per cent of the deaths. The mortality in the group of cases admitted on the fourth day of illness was 14 per cent; the combined five, six, and seven day group showed a mortality of 10 per cent; whereas the group with symptoms lasting more than a week, gave no mortality at all, although they were 8 in number. Certain facts are evident: (1) the safest period for operative intervention is during the first twenty-four hours of the attack; (2) a sharp and sustained rise in mortality occurs in the cases where the duration of the attack continues beyond forty-eight hours and (3) after a weeks duration, localization of the infection oftentimes has advanced to such a degree that operation can be performed with a satisfactorily low mortality. On the other hand, there is the group of neglected or ill treated cases seen late in the course of the disease, which shows very widespread and progressive peritonitis. Here the mortality is astoundingly high. Thus it is apparent that the great problem in appendicitis lies in the management of those cases which have been ill forty-eight hours or longer, and which come to the hospital extremely sick with signs frequently of a spreading peritonitis.

The importance of time as a factor in regulating the mortality in acute appendicitis was emphasized by Bower in his recent survey of appendicitis in Philadelphia. There, during a period of five years, in response to a vigorous program of public education, he found that the average time interval of cases admitted to operation was reduced no less than six hours. Coincidentally, clean cases in the series rose from 57 per cent to 73 per cent, an actual increase of 29 per cent. Most important, the mortality during that time showed a very gratifying drop from 5.9 per cent to 3.5 per cent, an actual decrease of 41 per cent.

The Metropolitan Life Insurance Company has published a map which shows the geographic distribution of the appendicitis death rate in the United States and Canada. The impressive thing about it is the very high mortality apparent in the Rocky Mountain states, and the only slightly lower mortality in the midwestern agricultural states. The altitude, climate, age, race and sex distribution of the population are set forth as possible reasons for this peculiar geographic distribution. However, careful checking of those factors proves that they have little or no influence on the high mortality reported in those states. It appears that the true explanation lies in the peculari status of those states with respect to available hospital facilities. Nearly all of them are sparsely populated and hospitals are few and far between. Obviously, people stricken with appendicitis must lose considerable time before they can obtain proper hospital treatment. It is probable that the physical condition of patients arriving for operation is far less favorable to recovery than is that of the average patient in other states, where distances are not so great.

It seems unlikely, therefore, that any great reduction of the mortality in appendicitis will occur over the United States as a whole until the element of time is better controlled through improved transporta-

tion and hospital facilities, especially in the more isolated sections of the country.

COMPARISON OF TWO YEARS

One of the objects of this survey was to study the information gained on the subject of appendicitis in this hospital during two periods having a fairly long interval between them, in order that, (1) a starting point might be established for future investigations into appendicitis at this hospital, and (2) in order to settle for this locality the question of whether the operative mortality of appendicitis is actually increasing or diminishing.

The statement is often carelessly made, that the operative mortality from appendicitis will show considerable variation from year to year, depending on the "virulence of the infections of the appendix." Just what is meant by that is not clear, unless reference is made to the incidence of Streptococcus hemolytic appendicitis, or the incidence of fulminating and early rupturing appendices, which occur during a given year. At the least, a small number of these is inevitable. It is quite true that the mortality in any given individual's rather short series of cases may fluctuate considerably from year to year, depending on whether he sees any, or several, severe cases. But in a series of any great size, covering years of time, it is doubtful whether there will be much variation, barring some radical departure in treatment. In support of this is Krech's series of 2207 acute cases from 14 hospitals in New York City in 1921, with a mortality of 7 per cent, and his series of 2334 cases, ten years later, treated in much the same manner, with a mortality of only 0.1 of 1 per cent less. The number of so-called catastrophes may affect very definitely the mortality of an individual's short series, but they too, are more or less constant in longer series. The factor above all others which affects mortality in appendicitis is the number and method of handling of the cases complicated by spreading peritonitis.

For convenience, the period of September, 1934, to September, 1935, was chosen for the recent year's series, and the corresponding months of 1929–1930, that is five years previous, for the older comparative series. The year 1934–1935 showed a mortality in acute appendicitis of 4.2 per cent, as compared with 6.2 per cent for 1929–1930. In other words, through improved surgery, better surgical judgment, and the results, such as they may be, of public enlightenment regarding the dangers of catharsis and procrastination in the treatment of abdominal pain, the mortality has dropped 32 per cent, and this in the face of a gradually increasing operating staff, comprised practically entirely of young and relatively inexperienced men. In 1929–1930 there was one death in the uncomplicated group, which was not true of the series of 1934–1935. The groups complicated by local peritonitis showed approximately the same mortalities for both years. Appendicitis with abscess showed a mortality increase of about 12 per cent in 1934–1935 over 1929–1930. It was in the treatment of the much debated and all important group of spreading or diffuse, or general peritonitis cases, that the great improvement in mortality occurred. In 1929–1930, 11 cases had diffuse peritonitis. Of these, 7 died, giving the astonishing mortality of 63.6 per cent. In 1934–1935, among 6 cases classified as diffuse peritonitis, there were 2 deaths,

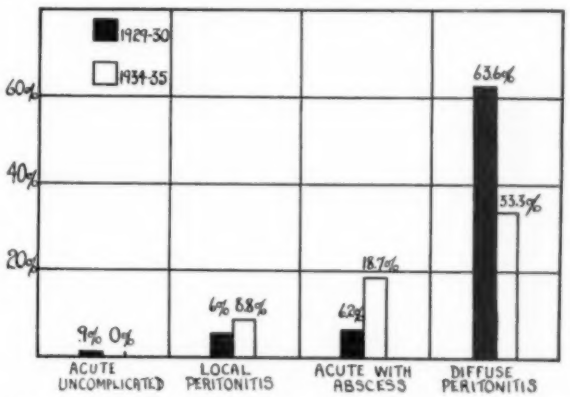

TABLE VII. Comparative mortality according to pathological types.

giving a mortality of 33.3 per cent. Improvement in mortality in practically all series, follows improvement in the results obtained in the complicated groups. The occasional mortality in uncomplicated appendicitis ordinarily runs from zero per cent to 1.5 per cent, and little improvement can be expected in this figure. Complicated appendicitis comprised 60 cases in 1929–1930 as compared with 56 cases in 1934–1935. The mortality in 1929–1930 in complicated cases was 16.6 per cent, as compared with 14.2 per cent in 1935, representing an actual decrease of 14.4 per cent.

Guthrie recently made the statement in a medical editorial that to include uncomplicated cases in the computation of mortality in acute appendicitis is to obscure the real issue. If results were reported only in terms of the complicated groups, he believes that the figures would be amazing. According to our own statistics, one person in every 6 admitted in 1929–1930 with appendicitis complicated by peritonitis, died. In 1934–1935, that ratio was lowered to one in 7.

The total number of acute cases operated in 1929–1930 was 176, and in 1934–1935 was 188. There were 12 more females than males in 1929–1930 and 20 more males than females in 1934–1935, so that the total incidence in the two series was practically the same. In 1929–1930 there was a marked difference in the mortality of the sexes, the male mortality being almost double the female. Males showed a mortality of 8.5 per cent, as compared to 4.3 per cent in the females. In 1934–1935, the mortality for the two sexes was practically the same. Thus the combined mortality for the two years was 5.9 per cent for males, and 4.5 per cent for females, a disproportion which is comparable to that observed in most other series.

Acute appendicitis is predominately a disease of the early decades of life. In the 1929–1930 series 79 per cent of the cases were thirty years of age or younger. In 1934–1935, 74 per cent were in the same age group.

In spite of the fact that the 1934–1935 series contained 12 more cases than the series of 1929–1930, drainage was performed eight times less often. In 1929–1930 one case complicated by peritonitis was not drained, while in 1934–1935 drainage was omitted in 4 such cases. In 1929–1930, 22 uncomplicated cases were drained, while in 1934–1935, 21 were drained. These figures reflect a very marked conservatism in the matter of abdominal drainage at this institution.

Data on the time interval, use of cathartics, previous attacks, and the type of incision are entirely too incomplete in this series to form the basis of any worthwhile conclusions. It is regretted that such widely discussed and controversial subjects have to be dismissed with an apology.

DISCUSSION OF DEATHS

It is true in surgery, as well as in the other branches of medicine, that one can learn a great deal from a study of the deaths. By this, it is not meant that deaths necessarily result from mistakes in diagnosis or treatment. However, by carefully reviewing the cases, noting the complications that occurred, observing the inadequacy of one form of treatment, or the value of another, considering other and perhaps better methods of treatment which might have been employed, the surgeon should become better able to cope with similar situations when he meets them again.

In 1929–1930 there were 11 deaths in the series of 176 acute cases. The uncomplicated group showed one death, which occurred in a twenty-five year old female, who died within twenty-four hours of operation with high temperature, very rapid pulse and convulsions. This is the only real catastrophe in that series, and was probably due to a cerebral accident, although autopsy was not performed. Of the remaining 10 deaths,

2 cases which on admission were thought to have localized peritonitis, developed general peritonitis after operation, and to delay operation for perhaps twenty-four or forty-eight hours, with the hope that rest, fluids, and supportive measures (after

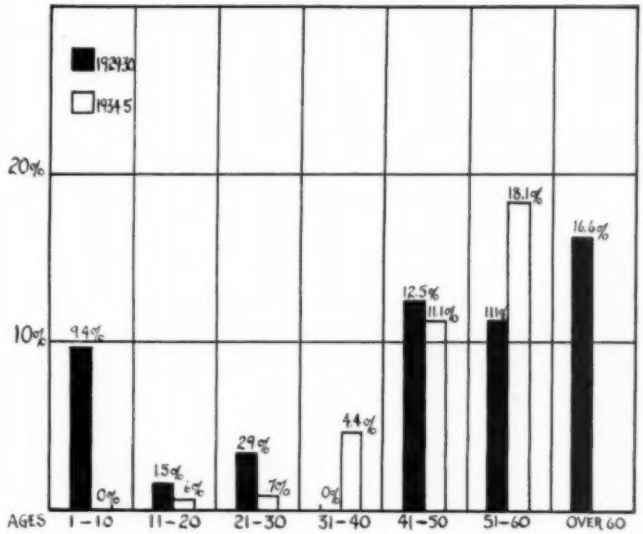

Table VIII. Comparative mortality according to age groups.

died. Another case had a Streptococcus hemolytic peritonitis. One case of appendicitis with local abscess developed general peritonitis after drainage, and died. There were 7 cases with spreading or diffuse or general peritonitis on admission, which died. Two of these cases were over 60 years of age and 3 were ten years of age or younger. These two age groups always offer great difficulty, both in the matter of prognosis and of treatment. The remaining 2 were seventeen and forty-eight years, respectively. All 7 cases died of general peritonitis after a varying number of days.

On closer inspection it is noteworthy that five of the deaths in this group occurred in patients which on admission showed unmistakable signs of a very severe infection, namely profound toxemia, rapid pulse and varying degrees of abdominal distention. All 5 died within the brief space of forty-eight hours following operation. This fact in itself causes one to pause and consider whether the best judgment was exercised in their treatment. It seems fair to question whether it might not have been better in these cases the method of Ochsner) might be of help in localizing their peritonitis so that abdominal drainage might be performed more safely and with a better chance of recovery. Three in this group were ten years of age or under, an age when the peritoneum and omentum are notoriously slow to localize spreading abdominal infection. A great difference in opinion exists regarding the wisdom of delayed treatment in these cases. However, the overwhelming nature of their infection, their poor surgical condition on admission, and the promptness with which they died following operation, make the critical observer wonder if conservative treatment would not have been the better judgment. Setting aside those 3 debatable cases, there are left 2, which it appears were ideally suited for delayed treatment. One presented an overwhelming diffuse peritonitis, the other a questionable local, but probably highly toxic, spreading peritonitis, both of which cases died within forty-eight hours of operation.

In the 1934–1935 series of 188 cases, the preoperative and postoperative data are more complete, and the group classifica-

tion probably more accurate. There were 8 deaths, 2 of which we have classified as catastrophes. One catastrophe, after a satisfactory convalescence from diffuse peritonitis for twelve days developed signs of cerebral accident, as manifested by convulsions and bloody spinal fluid, and died on her fifteenth postoperative day. The other, a case of early ruptured appendicitis developed violent ether convulsions during operation, and died of cardiac failure sixty hours after operation. Autopsy showed practically no sign of peritoneal infection. Of the remaining 6 deaths, 2 occurred in cases having Streptococcus hemolytic peritonitis. Both died within forty-eight hours of operation. One of these cases revealed at operation spreading peritonitis coexisting with a phlegmonous infection of the abdominal wall. The other, a thirteen year old male had lobar pneumonia and pleuritic effusion of streptococcic origin, complicated by a positive blood stream. On the fifth day of his illness, he developed right sided abdominal pain and distention. He was operated four days later after gradual progression of his abdominal complaints. A thin peritoneal fluid and inflammatory adhesions were encountered in the region of the appendix. The appendix was not found. Culture of the abdominal fluid produced hemolytic streptococcus. The patient died within twenty-four hours of operation. It seems reasonable to question whether this might not have been a peritoneal infection secondary to the pneumonia and pleurisy, but in as much as it was thought by the operator to be of appendiceal origin, it is so considered here. There was one case of appendiceal abscess of seven days duration in a fifty-two year old male, who died of general peritonitis ten days after drainage of the abscess.

Three cases are left, having histories of forty-eight to seventy-two hours duration. All died of general peritonitis following operation. One was a very obese male of twenty-nine years of age, who gave a history of drastic catharsis. At operation, the appendix could not be found although search was made for it among adhesions. Drainage was instituted. The postoperative course was not good. He developed general peritonitis and a fecal fistula, dying on the twenty-third day after operation. The final 2 cases are offered as probably better suited to delayed than radical treatment. One was a forty-six year old female, having an abdominal history of more than forty-eight hours duration. On admission her pulse was 120, she was very toxic, and showed signs of a spreading peritonitis. She was operated soon after admission and died of general peritonitis on her fifth day after operation. The other, a fifty-eight year old female, with a three day abdominal complaint, showed a profound toxemia on admission, with a pulse of 140, which was weak and thready. She was also operated shortly after admission and died twenty-four hours later.

It thus appears that in the series of 364 cases of acute appendicitis, there were 4 cases with peritonitis whose duration of illness and general clinical picture on admission placed them more or less unequivocally in the group which many surgeons now believe is properly treated by temporarily substituting physiological rest and general supportive measures for operation in the hope that localization of the abdominal infection may occur so that peritoneal drainage may be performed later with a better chance of life expectancy. This ratio of approximately one case in 90, warranting delayed treatment, corresponds to the experience of many surgeons, who occasionally make use of this procedure. Its application, however, can be more general. The extensive series of cases reported now and then by its proponents can be explained by its rather routine use in many of the cases of early peritonitis, which in the past have been treated by immediate operation in the hands of most surgeons.

THE TREATMENT OF SPREADING PERITONITIS

Any consideration of the treatment of appendicitis complicated by spreading peritonitis resolves itself into a discussion of the relative merits of immediate operation as opposed to delayed operation in that type of case. There is no problem in surgery that calls for greater evidence of experience and sound judgment than the proper management of ruptured appendicitis. Other things being equal, wisdom in surgery usually increases with a man's experience, but Bower's survey in Philadelphia showed that a surgeon could spend decades managing spreading peritonitis, and still have a mortality of 65 per cent. Usually it requires experience and judgment developed through many years of practice to know when to operate in the complicated case, and how much to do. Bower's survey, which covered more than 18,000 cases of acute appendicitis, enabled him to draw certain conclusions. One was that the average surgeon can manage patients with appendicitis complicated by spreading peritonitis fairly successfully, providing they have not had laxatives. If a laxative has been administered, they have only one chance in 7 of recovery. Not only is the peritoneal soiling more marked in cases which have been given cathartics with rupture of the organ, but what is perhaps more important, the intestinal tract has been stimulated to hyperperistalsis. This may result, not only in mechanical spread of the peritoneal infection through movements of the intestinal loops, but also may prevent the formation of peritoneal adhesions, which serve to wall off the infection and prevent its further dissemination. Furthermore, about one case in 100 perforates or develops general peritonitis in less than twelve hours. Without exception patients developing perforation with fulminating peritonitis before twelve hours have been given laxatives.

The practice at this hospital has been to operate "early and often" in both the uncomplicated and complicated cases. This procedure has resulted in a mortality for 248 uncomplicated cases of 0.4 per cent, a figure that is eminently satisfactory. However, in 116 cases, complicated by peritonitis, there were 18 deaths, giving the alarming mortality of 15.5 per cent. Of this group local peritonitis showed a mortality of 7.5 per cent, appendicitis with abscess, the high mortality of 12.5 per cent, and diffuse peritonitis the appalling mortality of 52.9 per cent. It is obvious from these figures that there is considerable room for improvement in the treatment of appendiceal abscess and in the management of general peritonitis at this hospital.

There is perhaps too great a tendency on the part of surgeons to make the treatment of appendix abscess an emergency. It is almost never a true emergency, unless under observation the mass gives signs of definite increase in size. Therefore, Nature should be given every opportunity to localize the infection, and to develop the blood and tissue resistance of the patient to their highest degree. The principal danger lies in peritoneal contamination, which can be obviated in some cases by extraperitoneal drainage, or by packing either to the region of the abscess, or to the parietal peritoneum and waiting for adhesions to form before opening into the abscess. In dubious cases there is probably greater danger in the drainage of an abscess which may not be well localized than in adherence to a policy of conservatism, which may allow it in a certain percentage of cases to rupture into the intestinal tract.

The proper treatment of spreading peritonitis probably requires more in the way of surgical judgment. This is the case that is ordinarily seen forty-eight hours or more after the onset of symptoms. The pulse is rapid, sometimes poor in quality, the patient is perspiring and distended, showing every evidence of marked intoxication. If operated before he has a chance to immunize himself, very often the patient will die. This is

the type of case which many surgeons are treating with conservatism, following a course of waiting hours or days if necessary for localization of the infection before operating. They take the stand that we do not now, or should not, interfere with Nature's localization of a skin or subcutaneous infection by making incisions over lymph channels that are clearly delineated by their redness. We give the patient a chance to immunize himself against the infection, and when his temperature and pulse rate have subsided and the lymphangitis has disappeared, with perhaps the formation of an abscess, a small opening is made and drainage instituted. The management of many cases of spreading peritonitis is identical. Here we cannot see what is taking place, but the manner in which the patient reacts tells us a great deal. A good rule to follow in these cases is "when in doubt, wait." It is well to take readings of the pulse and temperature every hour and leucocyte counts every four hours in order to determine the course and management of the case.

There are two schools in the treatment of perforated cases seen after the second day of the disease. The radical school, which the procedure at this hospital may be said to represent, believes that operation should be performed at once, irrespective of the condition of the patient or the symptoms present. The appendix is quickly removed, if accessible, and an accepted postoperative plan instituted for the treatment of the peritonitis. Opposed to this is the conservative school, which believes that it is best to stand by in the presence of signs of a peritonitis as shown by a fever, rapid pulse rate, and distention, when there are no signs of localization, and to assist the patient in every way possible to kill off the infection and to encourage localization. This is done by the use of Fowler's position, continuous gastric lavage, heat or cold to the abdomen, morphine in large doses, and parenteral injection of fluids, in order to render nourishment and maintain the chloride level of the plasma. When a degree of localization has occurred, as evidenced by improvement in the pulse and temperature, and softening of the abdomen, with perhaps formation of a palpable mass in the appendix region, operative intervention is indicated, and a procedure is employed which is simple, affective, and least taxing to the precarious resistance of the patient. When operation is undertaken in these cases, it is important to remember that the pathological organ need not necessarily be removed, nor the entire pathological zone be subjected to surgical exposure. Surgical intervention in peritonitis, in the words of Heyd, rests upon the simple procedure of instituting drainage to the site of the infection, and to the relief of the concomitant intestinal obstruction.

Guthrie says that misunderstanding and misapplication of the principles of the Ochsner treatment have brought the plan into disrepute in the minds of many. However, he has used it for years in this stage of appendicitis, and is confident that in his hands at least, it saves more lives than the more radical measures which he has tried and since abandoned. Exception is made in the case of one group. Infants, with their rudimentary omentum and delicate abdominal structures, cannot wall off infections adequately, and therefore radical procedures are almost always advisable with them.

It is interesting to compare the mortality of 15.5 per cent, representing the result of radical treatment in complicated appendicitis at this institution, with the mortality obtained by radical measures elsewhere, as well as with the results obtained in series where delayed operation was practiced.

Gile and Bowler confined themselves almost entirely to radical measures in the treatment of their series of 235 complicated appendices. There were 16 deaths, giving a mortality of 6.8 per cent, which is very creditable. Operation was delayed from

one to three days for preoperative preparation in 12 cases which had abscess formation.

Remarkable results in the radical treatment of ruptured appendicitis complicated by spreading peritonitis are reported by Gamble. His 129 cases showed 2 deaths, or a mortality of 1.5 per cent. The "open method of operation" which he employs is worthy of consideration. This treatment is based upon the assumptions that (1) in 98 per cent of the cases of peritonitis, anaerobic organisms are present; (2) that with the removal of the appendix the peritoneum is able to overcome any infection; (3) that infection of the abdominal walls plays a more important role in the causation of fatal sequela than any other factor; (4) that the closure, layer by layer, of the abdominal wall, provides both the culture medium and an excellent incubator for the growth of anaerobic organisms; and (5) that it is contrary to all surgical principles to close a fresh wound which has just been bathed freely in pus. His procedure consists in abdominal drainage, with closure of the peritoneum to the drains, reinforcement of the abdominal wall, with mattress sutures placed down to the peritoneum, washing of the wound with ether, and packing of the wound with gauze saturated with antiseptic. Position on the abdomen is maintained for twenty-four hours following which the wound is dressed and the pack removed. The wound is then left open and exposed under a tent which has a forty candle power electric light. Additional measures, such as nasal tubes, parenteral fluids, gas-gangrene serum, etc., are administered as indicated. By adherence to this practice, he has lowered his mortality during the past ten years in this difficult group from 9 per cent to 1.5 per cent, a figure for spreading peritonitis which is truly phenomenal.

Shipley and Bailey reported 66 complicated cases in which very radical procedures were used. The cases were operated immediately through the McBurney incision. After removal of the appendix a painstaking toilette of the peritoneal cavity was performed followed by irrigation of the pelvis with saline solution. Most of the cases of beginning peritonitis were not drained. There were 4 deaths in the series, giving a mortality of 6 per cent. Reporting later on his results covering a period of three and one-half years, where drainage was limited almost entirely to the late diffuse cases and cases of local abscess, Shipley reported 10 deaths in 121 complicated cases, a total mortality of 8.3 per cent.

Cottis and Ingham, writing on the non-drainage of peritonitis complicating appendicitis, reported a mortality of 12.6 per cent in a series of 95 cases.

Kennedy speaks strongly in defense of radical measures. He uses the coffer dam gauze drain instead of tube drains. Inserting this wall of gauze necessitates the breaking down of many protective adhesions. He reports a mortality of between 5 and 6 per cent in peritonitis cases.

These mortality figures ranging from 1.5 per cent to more than 12 per cent in series of complicated cases of appendicitis represent some of the best results obtained by radical treatment. Data are not plentiful on the subject of conservative treatment or delayed operation, partly because conscientious performance of the treatment requires much time and a great deal of careful supervision on the part of the surgeon, and because its periodic use develops only an occasional series of any great length.

It should be understood that the principle of delayed operation in the strict sense applies only to the case of ruptured appendicitis in which the inflammatory process has existed for more than forty-eight hours and where definite signs of spread of infection are present as evidenced by intoxication, rapid pulse rate and abdominal distention. The weight of surgical opinion still stands by the belief that the early ruptured appendix should be removed at once.

Coller and Potter report an interesting series of 85 cases, all of which were diagnosed "spreading peritonitis" on admission, and were treated by delayed operation. In reality, they were practically all diffuse, with a few cases of local peritonitis. Of this number, 8 patients died, giving a mortality of 9.4 per cent. Of the 77 survivors, 29 or 37 per cent grew better and were discharged on an average of thirteen days after admission. No abscess developed in these cases. In 48 patients, or 62 per cent, abscess developed. Drainage was instituted in these cases and the appendix was removed if it was easily accessible. Of significance was the fact, that in patients under sixteen years of age treated by this method, the mortality was 12.5 per cent, a figure which compares very favorably with the results obtained in this age group by other methods. This result was better than they had previously obtained in the same group, using immediate operation.

To Guerry, we are indebted for some fine comparative statistics. In 1926 he treated 123 cases of spreading peritonitis by deferred operation, with 2 deaths, a mortality of 1.6 per cent. At the same time 85 cases of diffuse peritonitis which were operated immediately, showed 7 deaths, or a mortality of 8.2 per cent. In 1932, 93 cases of diffuse peritonitis were operated immediately, with 10 deaths, a 10.7 per cent mortality, while 128 cases of diffuse peritonitis treated by deferred operation showed 2 deaths, or a mortality of 1.5 per cent.

Bailey reported his results, covering an experience of more than two years with the Ochsner-Sherren treatment. His statistics showed 242 cases operated within six hours of admission (mostly uncomplicated, but including 24 cases with general or severe peritonitis) with 4 deaths, a mortality of 1.6 per cent. At the same time 73 cases of severe, diffuse peritonitis treated by delayed procedure had one death, the remarkable mortality of 1.4 per cent.

Shute had a fairly representative experience with cecal drainage in treating 117 complicated cases of appendicitis. His final conclusions regarding cecal drainage are that the mortality is higher by this method among cases of localized peritonitis; that cases of appendiceal abscess require only incision and drainage anyway; and that in the remaining group, that is, diffuse peritonitis, he is convinced after studying the literature that cecostomy has far less to offer the patient than the procedure of delayed operation.

One may gather from the foregoing that delayed operation in the hands of some men has a rather wide application, and moreover, that conservatism in the treatment of appendicitis with peritonitis may carry with it an amazingly low mortality. The usual application of the method, however, and what is considered its greatest therapeutic possibility, is in the treatment of the neglected, extremely toxic case of peritonitis, whose natural immunity has been practically exhausted, and whose acquired immunity has not yet been developed.

BACTERIOPHAGE

Prompted by the more or less general interest that has been manifested in the use of bacteriophage in the treatment of various infections, a short series of complicated appendices was studied during the period of September, 1934, to September, 1935, in an attempt to determine something of the value of this agent in the treatment of ruptured appendicitis.

The preparation* used was a pooled lysate and filtrate of "colon bacillus, staphyloccus, different strains of streptococcus, and the types of pneumococcus." The finished product contained specific lytic or bacteriphagic, as well as antigenic and non-specific properties. The lysate was contained in 20 c.c. ampules.

The procedure usually followed was to empty an ampule of the preparation into the abdominal cavity, or introduce it through a drainage tube or catheter after the closure of the peritoneum had been completed. The dosage used was 20 c.c. at

* Cololysate Combined—product of Ely Lilly & Company.

the time of operation and 10 or 20 c.c. the following day. In one case it was used hypodermically, starting with 0.5 c.c. and increasing the dose to 1 or 2 c.c. on successive days.

Good results have been reported in short series where this "polyphage" has been used. At the St. Louis City Hospital, among 49 cases of perforated appendicitis treated with bacteriophage there were 5 deaths, a mortality of 10.2 per cent, while in 46 controlled cases in which the phage was not used, there were 7 deaths, a mortality of 14.5 per cent. Of the former group the combined or polyphage was used in only the last 20 cases, a monovalent colon bacillus phage, having been employed at first. Among the 20 cases, there was one death, a mortality of 5 per cent, as compared to 3 deaths in the control group of 15 cases where the mortality was 20 per cent.

Ruddell, Sicks and Loomis published their results with combined bacteriophage which they used in a series of 27 cases of peritonitis of appendiceal origin. The total mortality for the series was 11.1 per cent, that for diffuse peritonitis 30 per cent. The impression of those men was that in comparison with previous similar cases, the mortality was lessened by the use of the lysate.

Beginning September, 1934, and continuing for a year, a combined bacteriophage was used on the surgical service of this institution in 15 of the complicated cases of appendicitis. There was a contemporary controlled series of 41 cases. Among the 15 cases treated there were 3 with localized abscess, with no death; 9 with local peritonitis, with no death; and 3 with diffuse peritonitis, with one death. This was the death previously mentioned, following ether convulsion, which showed at autopsy almost no evidence whatever of peritonitis. The mortality in this short series was 6.6. per cent, as compared with 16.9 per cent for the 41 cases of the controlled series. It is of interest that this variation in mortality occurred despite the fact that the group treated by bacteriophage showed a higher proportion of diffuse peritonitis cases. The ratio was 1 diffuse to 3 cases of local peritonitis, as compared to 1 diffuse to 5 cases of local peritonitis in the control series.

In their enthusiasm, some who have used bacteriophage have probably attributed to it more credit than it actually deserves. However, it seems safe to say, and the statistics available lend confirmation, that this agent may become established as a worthwhile and valuable adjunct in the treatment of peritonitis from ruptured appendicitis.

SUMMARY

Certain conclusions may be drawn from this mass of information.

1. The operative mortality for acute appendicitis at this hospital, under a regime of radical treatment as practiced by a sizeable group of surgeons employing varied techniques, was 4.2 per cent during the year 1934–1935. This represents a marked reduction in the mortality of five years previously. While this mortality in no sense is comparable to that reported by certain clinics, better hospitals, and in various individual series, it nevertheless represents a rather respectable effort to cope with a problem of increasing difficulty.

2. Inadequate hospital records prevented an evaluation of the importance which the factors of time and cathartics may have played in effecting this lowered mortality, but it is certain that better surgical judgment and improved surgery were largely instrumental in producing the marked drop in mortality which occurred in the group of cases complicated by peritonitis.

3. The mortality in acute uncomplicated appendicitis at this institution is creditable and probably almost irreducible, but the treatment of appendicitis with abscess still shows a mortality which is much too high.

4. A short experience with bacteriophage as an adjuvant in the treatment of ruptured appendicitis is reported. No apology is made for the brevity of the series since the cases are considered to be representative. However, it is appreciated that only

through trial in a reasonably long series of cases can a true estimate of its value be reached.

It is likely that no one operative procedure will effect a very material drop in mortality among the severe cases of appendicitis. Improved mortality in all probability will result largely from the exercise of better judgment as to when to operate and how much to do. However, three points in operative procedure do stand out as worthy of particular consideration in any plan for reducing operative mortality in appendicitis.

1. The weight of evidence seems to indicate that in the very small percentage of cases where a severe spreading peritonitis complicates the picture, a regime of conservative treatment and temporary delay in operation offers the patient the best chance of recovery.

2. Controlled series seem to show that the use of the McBurney incision or its modifications favors a lowered mortality by confining operative manipulations to a very limited area of the abdomen, thus helping to keep the peritoneal infection localized.

3. In an admittedly short series of cases, bacteriophage has shown that it is worthy of further investigation in the treatment of cases complicated by peritonitis.

The experience has been that improvement in the hospital management of acute appendicitis is capable of producing a marked lowering in the mortality rate of this disease. It appears probable, however, that any considerable mortality drop will occur only when the public is thoroughly enlightened regarding the dangers of catharsis and of procrastination in the treatment of abdominal pain, and when routine early hospitalization of these cases will make possible removal of the appendix at a time when the operation carries with it, in experienced hands, a mortality which is almost negligible.

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PRIMARY EPITHELIAL TUMORS OF RENAL PELVIS*

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PRIMARY tumors of the renal pelvis arise apart from the kidney and are of very rare occurrence. Albarran collected 42 cases and Israel found only 2 primary growths of the renal pelvis in 68 tumors of the kidney. Until the present approximately 225 such cases have been reported in the literature and this report adds 5 additional cases. The incidence of these neoplasms has been variously reported as from 1 to 5 per cent. Verne Hunt, in a recent article on the same subject, estimated the ratio of pelvic tumors to renal parenchyma tumors as 1 to 14. At the Long Island College Hospital only 3 cases were found in a search of the records as far back as 1916. Secondary growths in this area are not uncommon and originate in the kidney parenchyma proper from which they extend and project into the pelvis. The infrequency of these tumors plus the difficulties confronting us in their accurate preoperative diagnosis and their treatment prompts us to report on these several cases which have come under our observation.

PATHOLOGY

The renal pelvis is subject to the same type of tumor lesions as the urinary bladder. According to Ewing, they may be classified as follows:

1. Papilloma
2. Papillary carcinoma (epidermoid carcinoma)
    a. Papillary epithelioma
    b. Squamous cell carcinoma
3. Alveolar carcinoma.

These are the more frequent forms and Cabot adds more uncommonly seen tumors of connective tissue or of embryonal origin, as the sarcoma and mixed tumor.

Papilloma. The benign papilloma is said to be the commonest type of primary tumor of the renal pelvis. It usually arises in the pelvis and metastasizes to the ureter and bladder by direct extension and implantation. These tumors are, as a rule, multiple and may be extremely numerous, covering the entire mucosa with fine vegetations. Occasionally, there is seen one large tumor with several small polyps scattered over the pelvis and calyces. The tumors are villous or wart-like, attached by a narrow pedicle and expand in a fan-like shape. Their great vascularity is a source of hemorrhage which is the chief symptom. Incrustation with salts and calculi are frequently seen. Various grades of hydronephrosis often result from partial occlusion, chiefly by ureteral transplants. The microscopic structure is typical and consists of elongated blood vessels covered by multiple layers of transitional epithelium. The tumor cells are cubical, cylindrical or elongated and spindle shaped, but the arrangement is orderly and the growth is confined to the mucous membrane. Round cell infiltration is found at the base of the tumor. The course of these tumors is usually benign but they may become malignant by invasion of the submucus layers, so that they should always be considered potentially malignant. Recurrences, after removal or from implantation in the wound, have occurred in a malignant form.

Papillary Epithelioma. This may be considered the malignant form of a simple papilloma. The transformation of a benign into a malignant papilloma has been first suggested and clarified by Albarran. Battle has demonstrated the malignant degeneration of a simple papilloma into a malignant tumor which recurred following the curettage of a previously benign lesion. Occasionally, focal malignant changes are seen in parts of a predominantly benign papil-

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loma, especially in the region of its pedicle. These tumors are more numerous and compact than the benign type. They appear as closely set, opaque, warty outgrowths which are usually found to cover a considerable area and may involve both the pelvis and ureter. Early, there is a tendency to encroachment on the submucous layers of the pelvic wall and frequently, into the parenchyma of the kidney, so that at times, it may be difficult to determine, in the gross specimen, whether the origin is renal or pelvic. These tumors rarely reach a large size as they cause so much bleeding that early removal is the rule. Microscopically, the papillary epithelioma shows overgrowth of the cell layers of the benign papilloma, atypical cells, and infiltration of the submucous tissues. In the infiltrating type, the papillary structure is soon lost and the growth becomes alveolar, diffuse or scirrhous.

Squamous Cell Carcinoma. This type of tumor is less common than the papillary form. These tumors are usually of a large size, although they may be small and discrete, involving a small portion of the pelvis. Its essential characteristics are infiltration into the pelvic wall with secondary involvement of the kidney and the ureter. Hydronephrosis and large cyst formation in the kidney are the rule and, combined with hematuria, make the diagnosis of the tumor at least presumptive. The microscopical appearance is that of any squamous cell carcinoma. Usually, the squamous changes are very pronounced and much of the growth may be composed of epithelial pearls many of which are hornified or calcific. The epithelium of the pelvis, according to Ewing, is capable of extensive epidermization as shown by the many reports of leukoplakia of the pelvic epithelium associated with calculi. The possibility of squamous cell carcinoma arising from a preceding leukoplakia is, therefore, suggested by some authors. This metamorphosis was noted by us in several recent cases in whom the kidney was exterpated for hydronephrosis, both infected and clean. Microscopic study of the pelvic epithelium revealed definite evidences of metaplasia.

Alveolar Carcinoma. Alveolar carcinoma of the renal pelvis may be considered to be an advanced and highly malignant form of papillary epithelioma, in which there is seen a loss of papillary structure and its replacement by a structure of alveolar, diffuse or scirrhus carcinoma. Ewing states that the early squamous pelvic tumors are described as flat and infiltrating rather than papillary. The advanced tumors are large, infiltrating and highly malignant.

METASTASIS

The simple papilloma, as a rule, is benign but must be considered potentially malignant because of its ability to spread into the kidney substance and also to the ureter and bladder by implantation. Recurrences of a papilloma are usually malignant. Distant metastases are extremely rare.

The papillary epithelioma is malignant and when not infiltrating, takes the course somewhat like the benign papilloma. If infiltrating changes occur, it becomes quite malignant, invading the kidney parenchyma and metastatizing to the local lymph glands, adrenal, liver, etc.

The squamous cell and alveolar carcinoma are highly malignant and disseminate early locally and at a distance.

ETIOLOGY

This disease seems to be one of adult life, most cases occurring in individuals between the ages of forty and seventy years, although they have been seen in the very young and the extreme aged. The condition is more often right sided and occurs somewhat more frequently in the male.

The origin of pelvic tumors is still conjecturable yet one cannot lose sight of the prominent part played by chronic irritation and infection over a long period. Both these factors, either together or alone, commonly result in the production of leucoplakial changes and other forms of

metaplasia of tissue and are to be considered distinctly cancerogenic. Albarran has suggested this causal theory. The not infrequent coincidental presence of renal calculi, producing long continued irritation, must be considered as a directly exciting and predisposing cause. Cabot found 5 of 18 cases of pelvic neoplasms associated with calculous disease. The majority of the tumors in these instances have been of the squamous cell type.

Whether in the life history of a new-growth there is a transition from normal through hyperplasia and metaplasia to neoplasia, is not definitely proved. Instances have been recorded in which has been noted metaplasia of the renal pelvis mucosa into skin with evidence of hornification. Melicow reports such a case associated with a large calculus which acted as the chronic irritant. We also have seen several cases recently showing similar changes. In one case a nephrectomy was performed in a female of sixty-one years for calculous pyonephrosis. The pelvic mucosa on examination was found to be stratified in type and on its surface strands of keratinized material were found. These alterations are the direct consequence of chronic irritation and infection and may be interpreted as an intermediary stage in the direction of the development of a neoplasm (Fig. 1).

The importance of congenital abnormalities in the production of these growths is problematical, although there have been reports of pelvic tumors in a horseshoe kidney. The rare instances of mixed tumors are without a doubt embryonic in origin.

SYMPTOMATOLOGY

The symptomatology of primary epithelial tumor of the renal pelvis is that of any renal neoplasm. The most frequent symptom is hematuria, at one time or another, reported in 70 to 90 per cent of the cases. The hematuria is often the first symptom, it is intermittent and painless, as a rule, gradually increasing in quantity and later giving rise to clots, the passing of which may be painful. The interval between attacks of bleeding may be as long as twenty years. In the benign tumors it

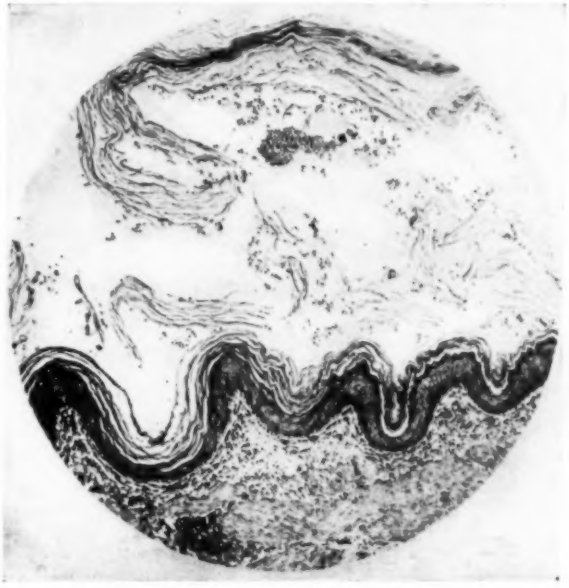

Fig. 1. Microscopic section of the pelvic mucosa of a kidney removed for a calculous pyonephrosis. Note the keratinization and stratification of the epithelium (45×).

may exist for several years before medical aid is sought. In some cases the cessation of bleeding is accompanied by an increase in the size of the tumor mass due to the blocking of the ureter by a blood clot and, according to Binney, this is to be regarded as an important diagnostic point. However, the presence of a renal mass, in these cases, is an infrequent finding. Thomson Walker states that the variations in size of the tumor mass in the case of pelvic tumors are much greater than are observed in new-growths of the kidney. Pain occurs as an early symptom in less than half the cases. It is dull in type and localized to the kidney region. Occasionally, a typical renal colic occurs due to the passage of a blood clot. The pain, as a rule, is due to an obstructive hydronephrosis.

At times one's attention will be drawn to the condition by the passage of portions of tumor tissue in the urine. This is especially so in instances of papilloma or papillary carcinoma. This finding, however, is rare

and is considered an extremely important sign.

Symptoms referable to the bladder are often noted in the form of frequency, nocturia and dysuria. This is due to the presence of blood clot in the bladder and incidental infection. In the later stages of the disease, loss of weight, anemia, cachexia and weakness develop and are prominent.

DIAGNOSIS

Since there are no characteristic symptoms of tumor of the renal pelvis, our findings obtained from the history and physical examination will lead us to suspect, in most cases, a parenchymal renal tumor. Cystoscopy and pyelography constitute very valuable adjuncts to our methods of diagnosis, for, the picture of a more or less dilated renal pelvis, within which one visualizes a filling defect, is fairly characteristic. Intermittent hydronephrosis or hematonephrosis, in the presence of hematuria and colicky pains, is extremely suggestive of a pelvic or ureteral neoplasm. The presence of a tumor transplant about one of the ureteral orifices or in the adjacent vesical wall, associated with these findings, points strongly to a neoplasm in the pelvis or ureter. The finding of tumor cells of the epithelial type in the urine, obtained by ureteral catheterization should be considered almost pathognomonic.

Taking everything into consideration, the diagnosis is difficult preoperatively. In most cases one would think first of a parenchymal tumor only to find a primary pelvic involvement upon surgical intervention. Thomson Walker states that the diagnosis has very seldomly been made before operation.

The diagnosis of the squamous cell type of carcinoma of the pelvis, is practically never made preoperatively since cystoscopic and pyelographic studies reveal no characteristic or suggestive data. In our case nephrectomy was performed for calculous pyonephrosis and subsequent examination of the specimen disclosed the associated malignant lesion.

In the differential diagnosis one must consider instances of massive hematuria of "unknown origin" which commonly show filling defects on pyelography due to the presence of a blood clot in the pelvis and also instances of incomplete filling of the pelvis because of insufficient dye. The pyelograms in these cases appear not unlike those of a papillary pelvic tumor. Also, it would be difficult to exclude a parenchymal renal tumor that had secondarily invaded the pelvis.

COURSE

The course of renal pelvis neoplasms varies with the type of tumor.

The simple papilloma, while benign, must be considered potentially malignant for two reasons: (1) because it commonly involves the ureter and bladder by implantation and, (2) because it may undergo malignant degeneration and its recurrences, after removal, are usually carcinomatous.

The papillary epithelioma is the least malignant of the malignant lesions of the renal pelvis. It offers the better prognosis. It, however, may spread to the ureter and bladder or invade the kidney substance. The degree of malignancy of these tumors is relatively low, grade 2 predominating in a series reported by Verne Hunt. This is supported by the ultimate results obtained. Metastases are said to occur late in the course of the disease and are not widespread. The grade of malignancy of both the primary tumor and the transplants or recurrences is usually of the same degree.

The sessile epithelioma or the squamous cell carcinoma is highly malignant, most of the tumors being of the grade 3 and 4 variety. These tumors invade the kidney substance and retroperitoneal tissues and also metastasize far and wide very early. All 8 patients with sessile epitheliomas of the renal pelvis reported by Hunt from the Mayo Clinic, died within a five year period; all except one lived less than six months.

Since alveolar carcinoma of the renal pelvis is considered an advanced and highly malignant type of papillary epithelioma,

its course is very short. They are all of the grade 4 type and metastasize early and extensively.

TREATMENT

In tumors of the renal pelvis, whatever the nature of the growth, surgery is indicated. The well known tendency of these neoplasms to progress by extension along the mucous membrane of the ureter and into the bladder, especially the papillary types, along with their high percentage of recurrences, makes it imperative that the treatment employed be of a radical nature. Complete nephroureterectomy, including the resection of portion of the bladder immediately adjacent to and surrounding the ureteral orifice, is the operation of choice and is recommended to insure the best prognosis. Any less radical procedure has proved, in the past, insufficient for a cure. We have found this operation easily executed employing the technique as described by Beer in cases of renal tuberculosis. Occasionally, where indicated, because of the patient's poor condition, it may be advisable to perform the operation in two stages, the nephrectomy first and later the ureterectomy. Simple nephrectomy is justifiable only as a palliative procedure for the painful symptoms in renal retention occurring in the inoperable cases.

It is wise to remember that even in the case of a simple papilloma, if a more conservative procedure is performed, there is a high incidence of recurrence in the ureteral stump or bladder or locally, sometimes is a state of marked malignancy.

The use of roentgen ray therapy offers very little hope in the treatment of this lesion judging from the reports in the literature. We, however, make it a rule to employ it postoperatively.

RESULTS

Five year cures are far and few between. Albarran, Giordano and Fenwick each have reported cases well five to six years after operation in frank malignant growths. In our series of 5 cases, I survived four years and then could not be traced further, 3 succumbed within six months, and in the fifth case, there is no record at all.

CASE REPORTS

Case 1. M. H., male seventy years of age, white, book dealer, was admitted to the urological service of the Kings County Hospital October, 12, 1935, complaining of hematuria of eighteen months duration.

Family and past personal histories were negative.

Present illness dates back to eighteen months ago when the patient first noted blood in his urine, varying in quantity from small clots to deep wine colored urine. There has been no pain referable to the kidneys nor any local vesical disturbance. There has been no weight loss but he has noted a progressively increasing weakness and lassitude. During the past six months he developed a cough which he attributes to smoking.

Physical examination revealed a large, elderly male with marked pallor but no other signs of importance. The kidneys were not palpable nor tender; the prostate was moderately enlarged, firm and nodular.

Cystoscopic examination under local anesthesia revealed a normal bladder but showed the bleeding originated in the left kidney. Function from both kidneys was within normal range and there was no infection. A left pyelogram revealed a moderate widening of the left ureter and left upper calyces, and a filling defect in the lower left ureter interpreted as possibly a tumor primary at this site (Fig. 2).

Opinion. Primary tumor of the left lower ureter.

A complete nephroureterectomy was performed under epidural anesthesia. The patient tolerated the operative insult well and reacted satisfactorily, but died suddenly on the twenty-sixth postoperative hour from a cardiac collapse.

Pathological Report. The gross specimen consists of a formalin-fixed kidney measuring 11 by 5 by 5 cms. The capsule strips readily. The capsular surface is roughened by innumerable, minute, slightly depressed scars and several large discrete scars and by a few ruptured cysts. The mucosa of the lower calyx is roughened by a slightly elevated papillary mass of gray tumor tissue measuring 2 cm. in diameter. The adjacent peripelvic fat is not

infiltrated by tumor and the parenchyma is free from tumor. The structural markings of the parenchyma are relatively undisturbed. The

A satisfactory history was not obtainable because of the patient's intellectual deficit concomitant with her age.

FIG. 2. Case 1. Pyelo-ureterogram showing the filling defect in the lower ureter.

FIG. 3. Case 1. Hemisected kidney showing the papillary tumor in the lower calyx, the ruptured cyst and the irregular renal margin.

mucosa of the pelvis and other calyces is normal save for a few petechial hemorrhages adjacent to the tumor. The ureter is not dilated and its mucosa is free from tumor (Fig. 3).

Microscopic. The pelvic mucosa is replaced by broad stratified papillary masses of atypical epithelial cells. The tumor cells are quite well differentiated and are fairly uniform in size, shape and staining qualities. They possess fairly large, round or oval nuclei, pale cytoplasm and distinct cytoplasmic borders. Mitotic figures are seldom seen. There is a moderate peripelvic fibrosis but the peripelvic tissues are not invaded. However, there is definite extension into a small area of renal parenchyma at the tip of a papilla, the invasion apparently being along the ducts of Bellini.

Diagnosis. Papillary epithelioma of the renal pelvis (Fig. 4).

CASE II. G. F., negress seventy years of age, was admitted to the urological service of the Kings County Hospital on October 15, 1935, complaining of difficult urination of three weeks duration, dysuria, urgency, strangury and intermittent hematuria of one year duration.

Physical examination revealed an aged, colored female, chronically ill and with a poor hemic component. The positive physical findings were right lower quadrant tenderness and a suprapubic lateral right rectus scar.

Cystoscopy disclosed a diffuse hemorrhagic cystitis. A pedunculated growth, about 2 cm. in diameter was noted on the left lateral wall of the bladder having all the characteristics of a benign papilloma. The ureters were catheterized, the drainage from the left catheter being markedly bloody and purulent. A left pyelogram showed a filling defect of the upper major calyces and a widening of the left ureter (Fig. 5). Intravenous urography revealed the same condition. The function of the right kidney was within normal limits. There was also noted a suggestion of a defect in the left lower quadrant of the bladder.

Opinion. Left renal tumor, possible papillary tumor of the pelvis; papilloma of the bladder.

In view of the patient's condition it was elected to perform a rapid nephrectomy first

and later a ureterectomy or simple fulguration of the vesical papilloma. The nephrectomy was performed on October 23 under epidural anesthesia, in the usual fashion, removing about two-thirds of the ureter. The patient died several days later of myocardial failure.

Pathological Report. The gross specimen consists of a formalin-fixed hemisection of a kidney measuring 11 by 5 by 2.5 cms. The ureter is not included. At one pole, the parenchyma is replaced by a firm, roughly hemispherical mass of gray white tissue measuring 4 cm. in diameter. The capsule covering this tissue strips readily, as it does over the renal parenchyma. The capsular surface is roughened by innumerable minute depressed scars and is mottled by scattered petechial hemorrhages. The pelvis and the dilated middle and upper calyces are occupied by a papillary mass of soft, gray white tissue which is in continuity with similar tissue replacing the parenchyma as noted. The parenchyma adjacent to the middle calyces is apparently not infiltrated by tumor but is atrophic, measuring a few millimeters in thickness. In the uninvolved portion of the kidney, the cortex averages 4 to 5 mm. in thickness and the medulla is somewhat narrowed in association with an increase in the adjacent peripelvic fat (Fig. 6).

Microscopic. The pelvic mucosa is replaced by broad stratified masses of atypical epithelial cells which form numerous projecting papillary structures supported by a vascular, delicate connective tissue stroma. The cells are moderately anaplastic and vary considerably in size and shape. The cells are pale but their cytoplasmic borders are distinct. Mitotic figures are common. The tumor in the parenchyma is essentially similar and almost completely replaces the parenchymal elements. A few minute groups of tumor cells are also found in the capsular tissue. The tumor cells do not manifest definite squamous characteristics, keratinization is not evident and no pearls are seen.

Diagnosis. Papillary epithelioma of the renal pelvis (Fig. 7).

Case III. N. A., white housewife, aged forty-nine years, was admitted to the urological department of the Long Island College Hospital, service of Dr. Augustus Harris, on October 17, 1930, complaining of pain in the right kidney area and upper right abdomen, frequency and nocturia.

Family history and past personal history were essentially negative with the exception of a single episode of right renal colic fifteen years

Fig. 4. Case 1. Microscopic section of tumor; note the invasion of the renal parenchyma at the tip of a papilla (15×).

ago at which time she passed two calculi, unassisted.

The present illness dates back to fifteen years ago when, following the renal colic the patient continued to experience a dull aching and burning pain over the right renal area accompanied by frequency of urination every one to two hours by day and 3 to 6 times at night. Four months ago, there was a marked aggravation of all the symptoms and she sought medical aid. At no time was any hematuria noted.

Physical examination revealed little of note excepting an easily palpable, markedly enlarged, tender right kidney.

Cystoscopic examination showed a generalized cystitis and marked infection and absent function in the right kidney. A plain *x*-ray plate revealed a stone cast of the pelvis and calyces. Pyelogram showed markedly dilated calyces and pelvis practically identical in shape to the calculus lying in it.

Diagnosis. Nephrolithiasis.

A very difficult nephrectomy was performed on October 20 under spinal nupercaine anesthesia from which the patient recovered without event. She left the hospital completely healed on the eighteenth postoperative day. After learning of the malignant nature of the condition, a full course of deep radiation

therapy was administered to the kidney area but the patient succumbed six months later of local and generalized metastases.

Fig. 5. Case 11. Pyelo-ureterogram.

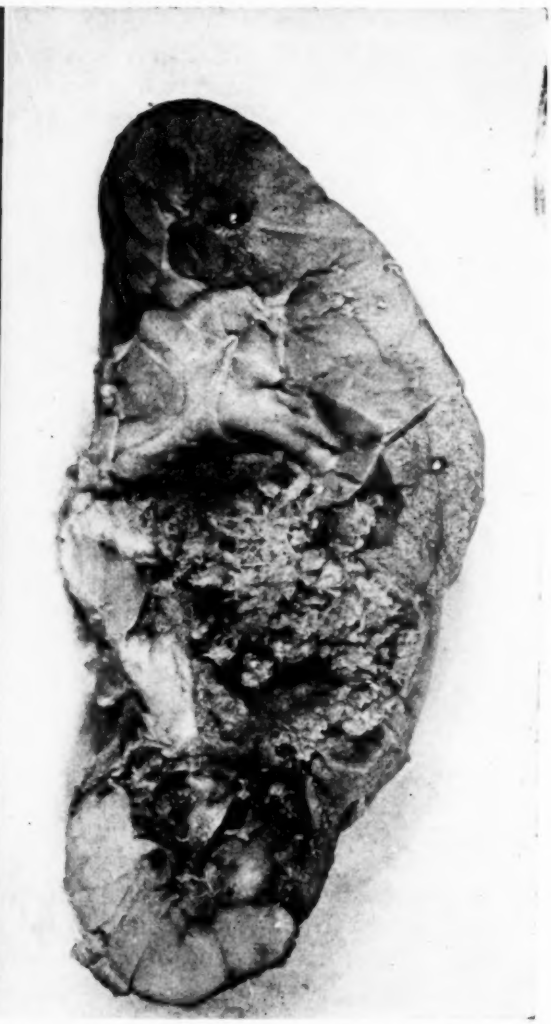

Fig. 6. Case 11. Hemisected kidney; note the papillary tumor filling the pelvis.

Pathological Report. The gross specimen consists of a kidney which has been removed with its surrounding fat. The latter is stripped from the organ with great difficulty and on one side infection has extended from the kidney into the perirenal fat. The kidney measures 10 cms. from pole to pole, 5 cms. in width at the hilus and 6 cms. in thickness. Its surface is irregularly lobulated by the presence of pouch-like dilatations of the cortex which are soft and apparently filled with fluid content. The cortical substance is greenish gray in color and apparently approaches necrosis and at widely diverging points numerous miliary abscesses are noted. On section, nothing recognizable as normal renal parenchyma remains, the entire substance of the organ being replaced by large cystic cavities, lined by a pyogenic membrane and all communicate with the renal pelvis. The latter is completely filled by a large hard but brittle stag-horn calculus. Several small stones occur in the cysts. The pelvis itself, is thickened, pearl-gray in color and covered by a pyogenic membrane. Attached to the specimen is an 8 cm. length of ureter which is somewhat thickened, dilated and lined by a thick mucosa which is injected and resembles granulation tissue.

Microscopic. Section from the cortex to the cystic pelvis includes a large area which is infiltrated by epidermoid carcinoma, squamous cell type. The tumor is arranged in alveoli of irregular size and shape separated by a moderately fibrotic stroma. Numerous epithelial

pearls are seen. The surrounding tissue shows little resemblance to renal tissue. Occasional glomeruli and scattered tubules are found in fibrotic connective tissue which is heavily infiltrated with subacute inflammatory cells.

Fig. 7. Case II. Microscopic section of the tumor; note the papillary structure.

Fig. 8. Case III. Microscopic section of the tumor; the squamous cell characteristics are prominent as are the pearls (40 ×).

Fig. 9. Case IV. Microscopic section of the tumor; note the papillary structure and the squamous cells and also pearl formation (30 ×).

Fig. 10. Case V. Microscopic section of the tumor.

Diagnosis. Nephrolithiasis, pyelonephritis; epidermoid carcinoma of the renal pelvis, squamous cell type (Fig. 8).

Case IV. G. R., white housewife, aged sixty years, was admitted to the urological department of the Long Island College Hospital, service of Dr. J. Sturdivan Read on July 6, 1927, complaining of pain in the left flank, a tumor mass in this area, hematuria and marked weakness.

Family history and past personal history were essentially negative.

The patient dated her present illness to seven months ago at which time she first noted blood in her urine, which has persisted intermittently since. This was associated with frequency of urination every two hours day and night. Pain

was first noted one week ago and has not left her, getting more severe and knife-like and is present constantly. She first became aware of

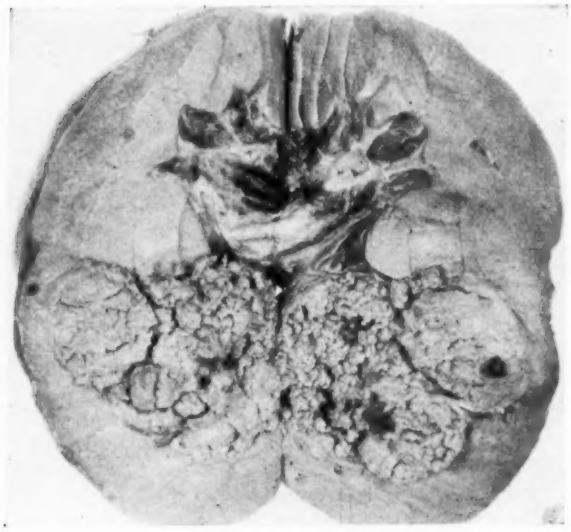

Fig. 11. Case v. Specimen showing plainly the papillary tumor invading the renal parenchyma.

the tumor mass six months ago; it has increased in size since and has become extremely tender.

Upon physical examination, it was noted that the patient was acutely ill and markedly anemic. The other pertinent findings were flatness on percussion in the left chest up to the seventh interspace, a large mass in the left upper abdomen, filling the entire kidney area and visible as a protrusion of the left loin. This mass is soft in consistency and tender.

Cystoscopy performed July 8, revealed a small papillomatous tumor protruding from the left ureteral orifice. There was a hydronephrotic drip from the left ureteral catheter and the urine from this side was markedly infected.

X-ray Study. The left kidney was not distinctly seen although there appears to be a large irregular kidney shadow with numerous areas of calcification, undoubtedly large stones, within the kidney pelvis. Below this kidney there is an oval shaped area of increased density which appears to be attached to the large kidney shadow. A left pyelogram shows a very large kidney pelvis with none of the usual markings. The ureter is large and dilated and there is a definite kink in the lower portion of its middle third.

Opinion. Calculous pyonephrosis with kink of the ureter.

A left nephrectomy was performed in the usual manner, the patient being transfused before and after operation. The postoperative course was uneventful, the patient was discharged on the sixteenth postoperative day.

This patient, four years later, was still well subjectively and objectively. Since then, it has been impossible to make contact with her or her family.

Pathological Report. Gross. The kidney is surrounded by a mass of fatty tissue. The capsule easily strips leaving an irregular mottled surface. The organ measures 13 by 7 by 7 cms. On section a large quantity of necrotic material is evacuated from several cavities. Papillary growths are found on the walls of the cysts and on the walls of the pelvis. Very little renal tissue remains.

Microscopic. The section shows the papillary carcinoma some areas of which contain malignant squamous cells and pearls (Fig. 9).

Opinion. Papillary carcinoma of the renal pelvis; calculous pyonephrosis.

Case v. The pertinent facts in this case are not known since the record could not be located. The gross specimen (Fig. 11), forms part of the museum of the urological department of the Long Island College Hospital and bears the name Mr. G. J., nephrectomy, 1920.

As can be seen, the hemisected kidney clearly demonstrates the large papillary tumor filling the entire pelvis and arising from its wall.

Microscopic sections made from a portion of the tumor mass show broad stratified papillary masses of atypical epithelial cells which are quite well differentiated and are of fairly uniform size, shape and staining qualities. Mitotic figures are few but present. There is moderate invasion of small areas of the renal parenchyma.

Diagnosis. Papillary epithelioma of the renal pelvis (Fig. 10).

CONCLUSIONS

Five cases of primary epithelial tumors of the renal pelvis are reported, 4 of which were of the papillary epithelioma type and one of the squamous cell type. Only one of these patients survived a four year period, the others all died within six months.

The pathology, etiology, symptomatology, diagnosis and treatment are discussed and the difficulties encountered in the accurate preoperative diagnosis and in

the treatment of these lesions are emphasized. The role played by chronic irritation and infection in the possible causation of these tumors is discussed and one case is reported showing transitional changes interpreted as premalignant. Two of our cases occurred in kidneys the seat of a calculous pyonephrosis.

We take this opportunity to thank Drs. J. Sturdivant Read and Augustus Harris for the privilege of including their cases in our report. We are grateful to Dr. Kenneth Strong for his assistance in reviewing the pathological sections.

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Anthropological accounts are replete with the abortifacient practices of primitive peoples. No one who has not surveyed these practices has any conception whatever of the gruesome, awful nature of the pain-inflicting processes by which the fetus was expelled, leading not infrequently to the ultimate death of the mother.

From—"Abortion" by Frederick J. Taussig, M.D.

We have seen cases in which an almost imperforate hymen has remained intact until the time of labor, and only ruptured when distended by the child's head.

From—"Williams Obstetrics" by Henricus J. Stander, M.D.

The occurrence of inertia in the first or in the second stage warrants the expectation that the usual contraction and relaxation of the uterus in the third stage will be lacking, and that there may be an atony. This frequently interferes with placental separation and expulsion and often results in postpartum hemorrhage.

From—"The Management of Obstetric Difficulties" by Paul Titus, M.D.

SURGICAL TREATMENT OF PUERPERAL SEPSIS*

A. F. LASH, M.D.

CHICAGO

ALL types of puerperal sepsis require the usual general medical measures, and although the majority respond favorably to these conservative procedures, there are certain types of puerperal sepsis which need surgical treatment. The recognition of these certain types of puerperal sepsis requiring surgical intervention may be difficult and at times not recognized until too late to expect a favorable outcome. It is the purpose of this paper to set forth those conditions requiring surgery and its reasons, as illustrated by specific selected cases.

The course of a puerperal infection may be progressive and, therefore, only by constant observation and repeated examinations can the changing pathological picture be appreciated. The pathological states warranting surgery are: beginning spreading peritonitis; abscesses of any part of the uterus, adnexa or pelvic structures; certain localized thrombophlebitis; and certain intrapartum infections.

General peritonitis is the most common cause of death in puerperal sepsis. In 51 autopsies of puerperal sepsis cases, performed by Dr. Jerry Kearns, Coroner's Physician at the Cook County Hospital, 47 per cent showed peritonitis as the cause of death, while in 100 cases of puerperal sepsis studied clinically and bacteriologically (by A. F. L.) of the 19 deaths, 9 were due to sterptococcal peritonitis. Many cases of peritonitis following cesarean section are overlooked because of the absence of abdominal distention and absence of generalized tenderness. Directing symptoms are the rapid pulse rate and toxemia which were present in 2 patients who died on the fifth and eighth postoperative day.

Pyrah and Oldfield in their excellent exposition of puerperal general peritonitis quote statistics which indicate that the incidence of general peritonitis in fatal cases of puerperal sepsis varies from 40 to 58 per cent. They discussed puerperal general peritonitis very thoroughly and reviewed the literature. It is evident that this condition is serious and once it has become general the prognosis is poor because there is no useful therapeutic procedure for it. The exception to this general statement is gonorrheal peritonitis which may heal spontaneously. However, before this advanced stage is reached or at the first indication of its beginning, much good may be done. First, the early diagnosis is based on the history in patients with metritis or walled-off inflammatory masses in the broad ligaments or adnexa developing symptoms of peritonitis or gradually taking a turn for the worse with such symptoms as chill, tachycardia, emesis, abdominal distention, rigidity, tenderness and diarrhea. All these symptoms may not appear at the same time but the significant ones are abdominal pain, tachycardia and distention. Pyrah and Olfield emphasized the triad of symptoms: rigor, abdominal pain and greatly increased pulse rate as indicating the onset of puerperal peritonitis.

With the diagnosis made of beginning spreading peritonitis, an immediate posterior colpotomy is performed under morphine-scopolamine analgesia, augmented by nitrous oxide-oxygen anesthesia if necessary. This operation is performed although there is no bulging in the cul-de-sac or fornices. The vagina is carefully prepared with a hot dilute (1–6000) potassium permanganate douche given by the operator. The posterior cul-de-sac is incised transversely 4–5 cm. and the peritoneal cavity is entered. Regardless of the presence or absence of exudate in the cavity,

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a fluffed iodoform gauze is inserted into the cavity, with a rubber T-tube if an exudate is present. The interesting finding in these cases has been the large amount, 200 c.c. or more, of exudate in the pelvic cavity although no bulging of the cul-de-sac or fornices was present. In only 4 instances of 27 colpotomies no or very slight amount (10 to 20 c.c.) of exudate was present. In only 11 instances was bulging present. Of the 16 cases without bulging, large amounts of exudate were obtained in 8. The absence of exudate does not mean that a colpotomy was not indicated because the gauze in the peritoneal cavity is a mechanical irritant, stimulating the peritoneum of the omentum and bowels to produce an exudate in order to wall-off or limit the infection to the pelvis. It is especially evident in the highly virulent spreading peritonitis that very little exudate is present, suggesting the action of aggressins against leucocytes, allowing for the extension and growth of the bacteria. Therefore, the reason for the colpotomy is not only to allow for drainage of pus if present but also to stimulate the localizing processes in the peritoneal cavity. In no instances have I felt that a colpotomy did any harm to a patient. Several case reports may further demonstrate the points made.

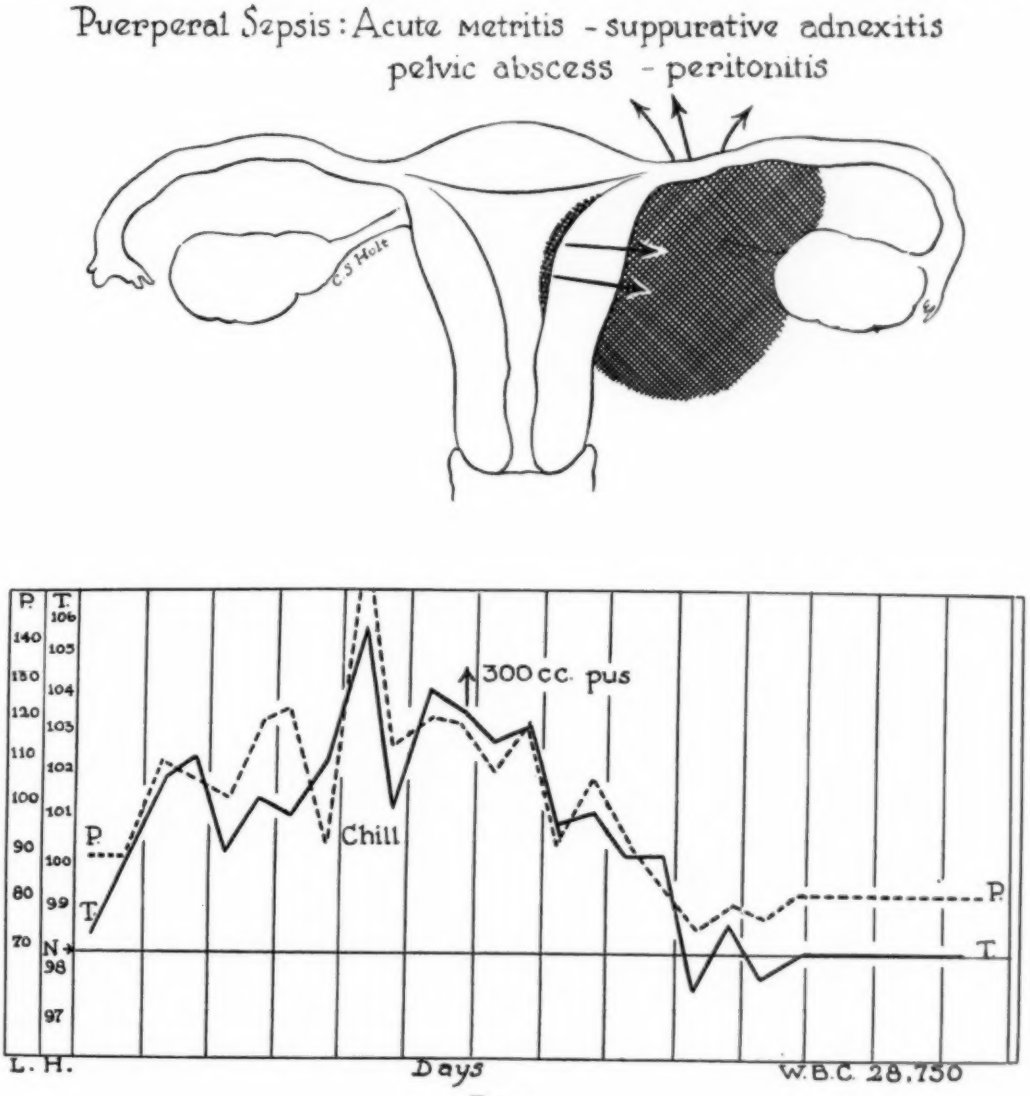


Fig. 1.

CASE REPORTS

CASE I. L. H., a twenty-four year old, white gravida III, para 0, entered the hospital because of vaginal bleeding and fever. She expelled an eight weeks fetus thirty-six hours before admission. She continued to have chills and fever; she passed pieces of placental tissue, following which the temperature would drop. On the fourteenth day in the hospital, the patient had a chill followed by a temperature of 105°E. and pulse rate of 160. Her abdomen became somewhat distended and tender, especially in the left lower quadrant. Her white blood count was 28,700 and the urinalysis revealed no pathological findings. Her condition remained about the same on the following day. Bimanual vaginal examination found the corpus somewhat enlarged, upright and fixed and a fullness in the left side of the pelvis with no bulging of the cul-de-sac. The diagnosis of puerperal sepsis, acute metritis, left broad ligament abscess with beginning spreading peritonitis was made (Fig. 1). A posterior colpotomy was done and about 300 c.c. of a yellow thick exudate was obtained. After the first twenty-four hours the temperature and pulse rate dropped and became normal on the fourth postoperative day.

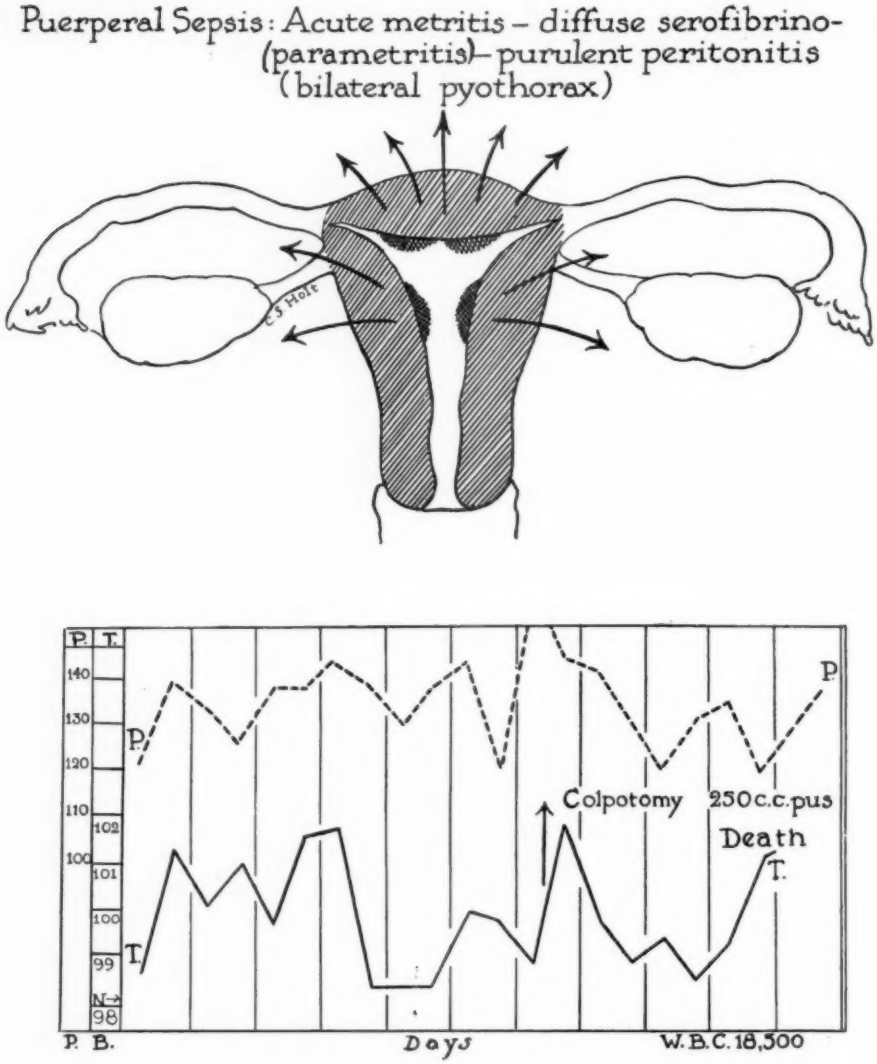


FIG. 2.

CASE II. P. B., a twenty-eight years old, white gravida IV, para III, came to the hospital because of cramps and swelling of the abdomen. She had induced an abortion with a catheter twelve days before admission, following which she began to have chills, fever, weakness, abdominal distention, nausea and vomiting. Her condition did not appear serious, since her

temperature was not high, varying from 98° to 101°F., but her pulse rate remained high and her abdomen was distended. Her white blood

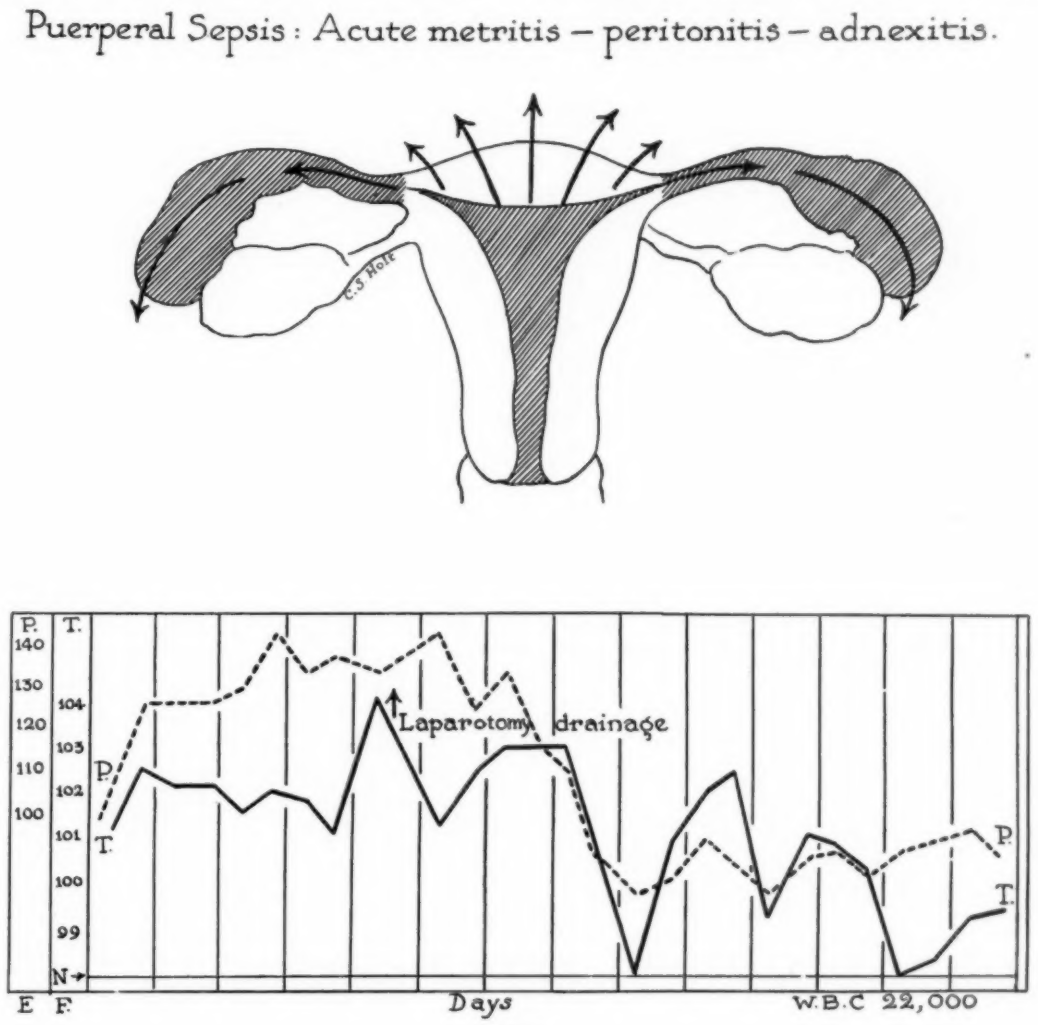


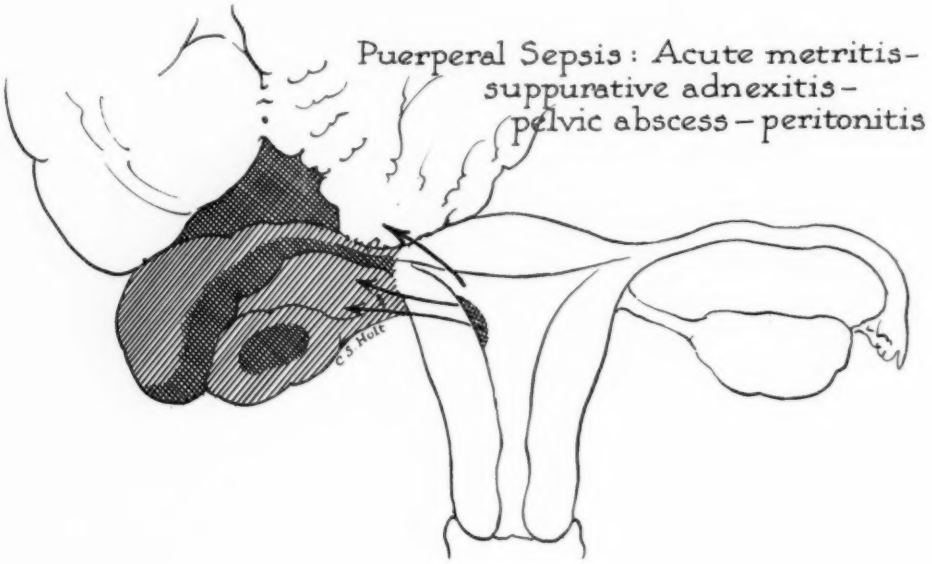
FIG. 3.

count rose to 18,500. On the sixth hospital day, a posterior colpotomy was done and about 250 c.c. of a thin, greenish-yellow exudate escaped. Although the patient appeared to improve the first day after the operation she gradually went downhill and died on the fourth postoperative day, with a terminal hypostatic pneumonia. The autopsy revealed infected placental remnants, acute metritis, diffuse serofibrinopurulent peritonitis, hyperemia and edema of the lungs and brain, and a bilateral pyothorax (Fig. 2).

There are instances, especially after full term deliveries, in which a colpotomy is not sufficient for drainage and then abdominal drainage becomes necessary. Although the English writers believe in primary abdominal drainage it is my contention that a simple colpotomy should be tried first since it may be sufficient. Only if the colpotomy proves inefficient, then abdominal drainage may be performed. Primary abdominal drainage is indicated when the abscess is high in the pelvic cavity and can not be reached through the cul-de-sac. A case report illustrating this follows:

CASE III. E. F., twenty-six years old, white, gravida IV, para II, entered the hospital because of abdominal pains and vaginal bleeding two days after a self-induced abortion with a skewer. Examination found the temperature 102.6°F. and pulse rate 128. The abdomen was somewhat distended, exquisitely tender in the lower half with no masses, but voluntary rigid-

ity, tympanitic throughout and absent peristaltic sounds. Vaginal examination found an enlarged, tender, soft, third degree retroflexed corpus uteri. The diagnosis was puerperal sepsis, acute metritis, probable perforated uterus and pelvic peritonitis. The blood picture showed white blood count of 22,000. The patient continued to complain of severe abdominal pain and since her condition was not improving, abdominal exploration was thought to be indicated. Four days after admission, on opening the abdomen, an acute metritis, adnexitis and diffuse fibronoserous peritonitis was found (Fig. 3). Culture of the exudate revealed a mixed growth of B. coli, staphylococcus albus and hemolytic streptoccocus. Drainage through lateral stab wounds and lower midline wound was instituted after the



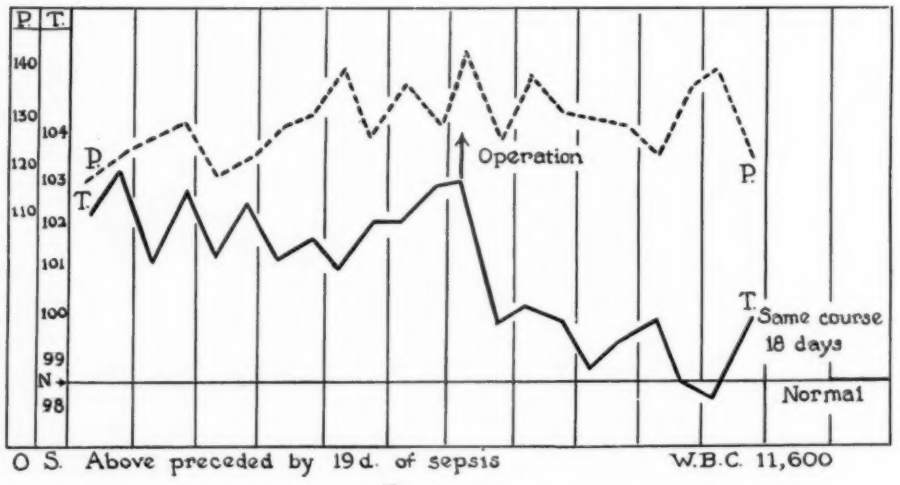


Fig. 4.

aspirated. A blood transfusion was also given. Although her general condition remained about the same, as indicated by temperature and pulse rate, she was comfortable and did not complain of pain. On the fourth postoperative day the temperature was normal and the pulse reached 72. However, there were recurrent rises in temperature. The wounds gradually stopped draining and healed. She went home thirty-eight days after operation. Subsequent examination found the corpus retroverted and fixed in the cul-de-sac with no adnexal masses. The patient's general condition was good.

The advantage of laparotomy is, better exploration and removal or drainage of tubo-ovarian abscesses. A case in point is that of the following:

Case IV. O. S., twenty-six years old, colored, gravida XI, para VII, aborted two days before admission to the hospital. The uterus was emptied with a dull curette and packed for twenty-four hours because of bleeding. A septic course followed. Since a mass about 10 to 12 cm. in diameter formed in the right lower quadrant, apparently well localized, the diagnosis of a tubo-ovarian abscess was made, twenty-four days after admission. At operation a large gangrenous tube was found, associated with a friable, infiltrated cecum, ovary and omentum to form an abscess (Fig. 4). Without disturbing the agglutinated structures above, the right tube and ovary were removed and two cigarette drains were introduced through a right stab wound, while the midline wound was closed. The high temperature and rapid pulse rate gradually subsided so that normal findings were present twenty days postoperatively and the patient was discharged on the twenty-eighth day.

When the clinical course indicates improvement, it is evident that an entire "hands off" attitude is assumed, regardless of the presence of inflammatory masses. The longer the waiting period, the greater the safety if surgery becomes necessary. However, the occurrence of pain and bowel obstruction may make surgery imperative as in the following instance:

Case V. M. S., an eighteen year old colored girl, developed an inflammatory pelvis, i.e., acute metritis, adnexitis, parametritis and pelvic peritonitis following an induced abortion. Her condition improved so that she thought she was able to go home. Two months later she returned because of lower abdominal pain, fever, and progressive constipation. Under palliative measures she became afebrile and improved in one month but the mass and pressure symptoms remained. Therefore a laparotomy was done and a large right tubo-ovarian abscess about 10 to 11 cm. in diameter was found. This ruptured during removal and pus spilled over the peritoneal cavity, culture of which revealed a hemolytic streptococcus. A right stab wound drain was made and the midline wound closed. With the exception of a stormy first twenty-four hours and a suture infection of the midline wound, the patient made an uneventful recovery and was discharged on the thirtieth postoperative day.

The importance of not operating an infected pelvis too soon is illustrated by the following case:

Case VI. E. A., white, thirty-five years old, para VI, gravida VII, came to the hospital, eighteen days after delivery because of pain in the lower abdomen. Pelvic examination found an acute metritis, right tubo-ovarian abscess and pelvic peritonitis. The patient was under observation for only two weeks, but because of pain and the presence of a mass, I decided to operate. At laparatomy a right suppurative salpingo-oophoritis (streptococcus hemolyticus) was found, the right adnexa was removed and drainage carried out through a right stab wound. The septic course persisted and the patient died on the eleventh postoperative day. Autopsy revealed a diffuse fibrinous peritonitis. Evidently the peritoneum had not become immunized to the hemolytic streptococcus as in the previous case.

Hysterectomy has been advised in the presence of a uterine abscess, sloughing fibroids (Berkeley and Bonney), infected ruptured uterus or in the absence of the source of peritonitis. Pyrah and Oldfield considered hysterectomy too formidable an operation in the presence of peritonitis and felt that even an abscess may be drained as well as a ruptured uterus, which should be repaired. J. F. Baldwin has been the chief champion of hysterectomy in certain puerperal infections which embraced the infections of the pelvic veins. There is still a difference of opinion as to whether a hysterectomy should be done. First, the difficulty encountered in the diagnosis of the condition requiring the hysterectomy such as necrosis of of a fibroid, suppurative metritis, localized abscess of the uterus, thrombophlebitis limited to the placental site, or even to the broad ligament as so often seen in an infected uterus with retained placenta. These conditions, although warranting a hysterectomy, may not be

recognized and appreciated until it is too late and almost impossible because of a parametrial involvement. Second, the removal of a uterus in a young woman may be warranted only as a life-saving operation. It has been my experience that retained placental tissue with infection followed by repeated chills and fever, and physical findings of non-fixed uterus and cord-like structures near the uterus presents the clinical picture of suppurative metritis and thrombophlebitis. The following cases illustrate this:

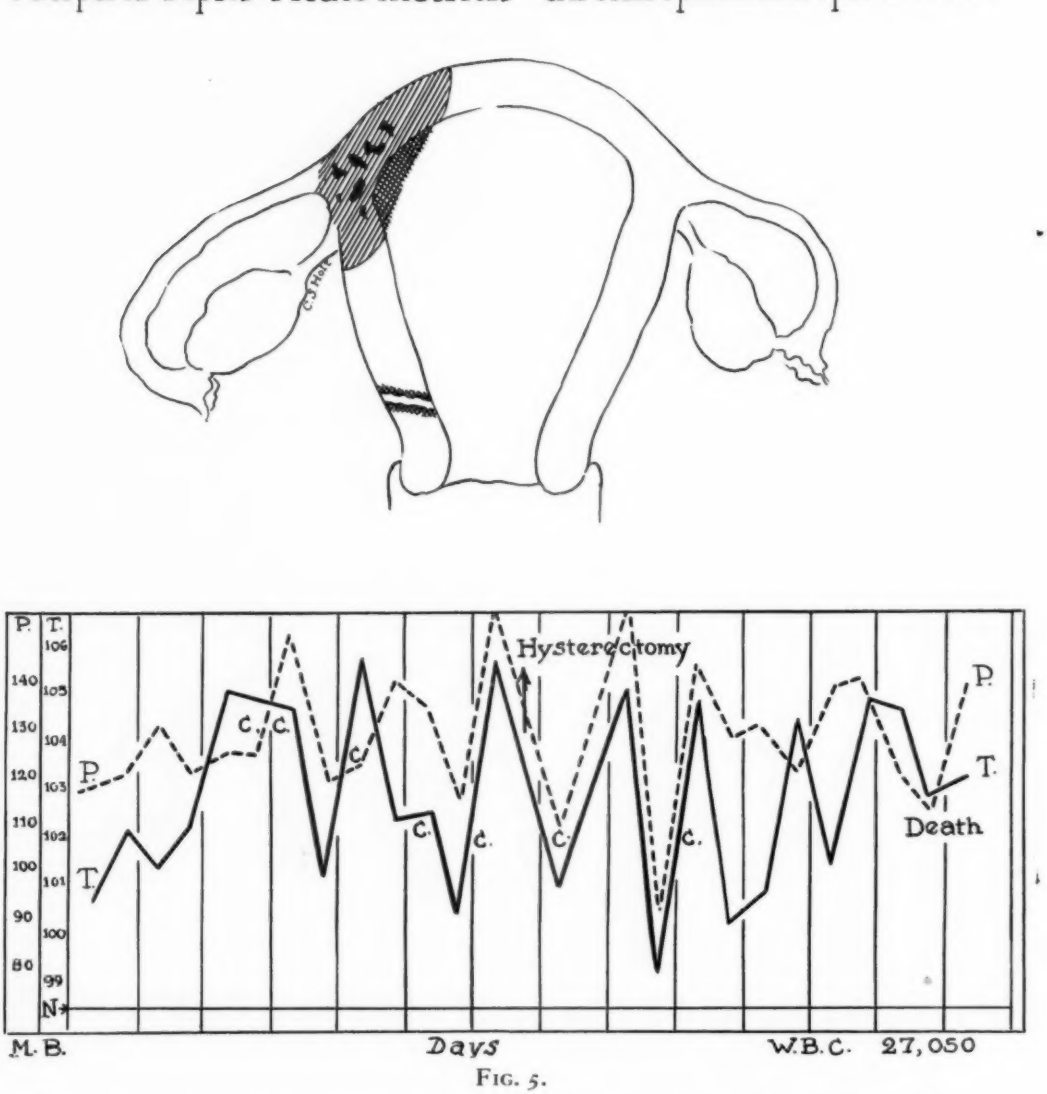


Fig. 5.

Case vii. M. B., twenty-nine years old, white, para v, had been in labor at home for four hours and had four vaginal examinations by her family physician before entering the hospital with a temperature of 101.6°F., and pulse rate of 124. She delivered spontaneously thirty-two hours later a full term, normal child. Twenty-four hours after delivery her temperature had gradually risen to 101.8°F., the pulse rate to 128. On the third postpartum day the patient had a chill, pulse rate 130 and temperature 102.4°F. The chills, with rises of temperature to 106°F. and pulse rate to 160, continued; on the eighth postpartum day the diagnosis of acute metritis, thrombophlebitis of uterine vessels was made. A subtotal hysterectomy was done and drainage through the vagina and the lower angle of the abdominal wall was instituted. A blood transfusion was given. The

uterus showed grossly a necrotic endometritis, laceration in the right lower uterine segment posteriorly, and thrombophlebitis of the vessels of the placental site (Fig. 5) Cultures of the uterus revealed a non-hemolytic streptococcus. The patient died on the eighth postoperative day, the septic course remaining unchanged.

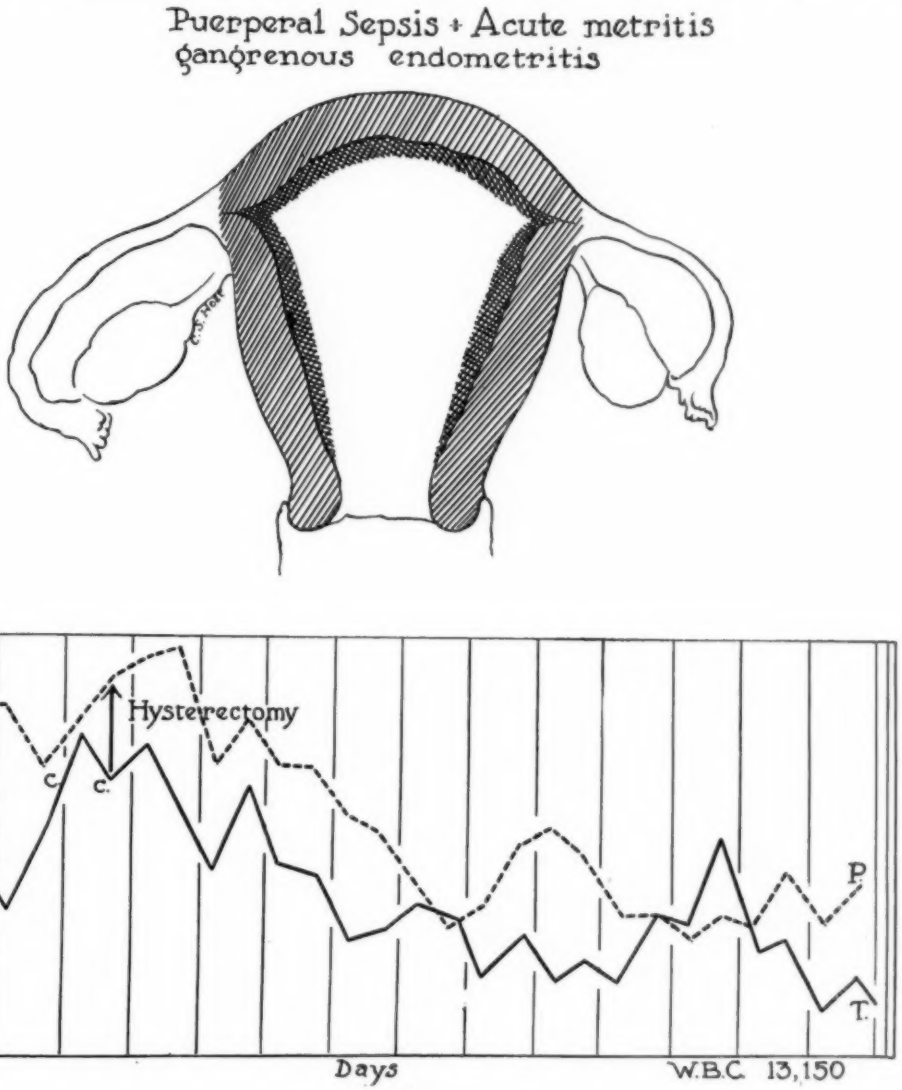


Fig. 6.

The patient was overwhelmed by an associated septicemia and I thought that interference was delayed too long.

Case viii. E. T., thirty-four years old, colored, para iii, gravida vii, delivered spontaneously a full term baby after twenty and one-half hours of labor but required manual removal of an adherent placenta and uterine packing because of postpartum hemorrhage. A blood transfusion of 850 c.c. was given. The temperature reached 102°F. and pulse rate 120 on the second postpartum day; on the third postpartum day the patient had chills with a repeated temperature of 104°F. and pulse rate 128. A diagnosis of retained placental remnants and acute metritis with thrombophlebitis was made. A subtotal hysterectomy was performed with drainage through the cervical stump and through the abdominal wall down to the peritoneum. The uterus showed grossly an acute gangrenous endometritis (Fig. 6), cultures from which grew B. coli and streptococcus viridans. The fever and pulse rate subsided by the sixth postoperative day and the patient made an uneventful recovery, leaving the hospital on the twentieth postoperative day.

Case ix. R. G., thirty-one years old, colored, para iv, gravida vi, entered the hospital because of vaginal bleeding and a seven months pregnancy. The diagnosis of placenta marginalis was made and a No. 8 Voorhees bag was inserted. The bag was removed at the end of twenty-four hours and a version performed which was followed shortly by delivery of a dead baby and then the placenta. A blood transfusion was given. On the second postpartum day the patient had a chill, a temperature rise to 103°F. and pulse rate 134. There was a profuse foul smelling, serosanguineous lochia. Since the chills and fever of 104.6°F. and pulse rate of 148 recurred and pelvic examination revealed a cord-like structure in the right parametrium and a gangrenous cervical canal, the diagnosis was made of puerperal sepsis, acute metritis, gangrenous cervicitis and thrombophlebitis of the right uterine vein due to a non-hemolytic streptococcus. On the fourth postpartum day a complete hysterectomy was done with gauze drainage through the vagina. The uterus showed grossly a gangrenous cervicitis and endometritis with bilateral thrombophlebitis of the uterine veins and parametritis (Fig. 7). The patient never rallied after the operation and died twenty-seven hours later with marked respiratory difficulty. Autopsy revealed a focal bronchopneumonia in the left lung and in the right lower lobe, localized fibrinopurulent peritonitis in the small pelvis, and the general findings of sepsis.

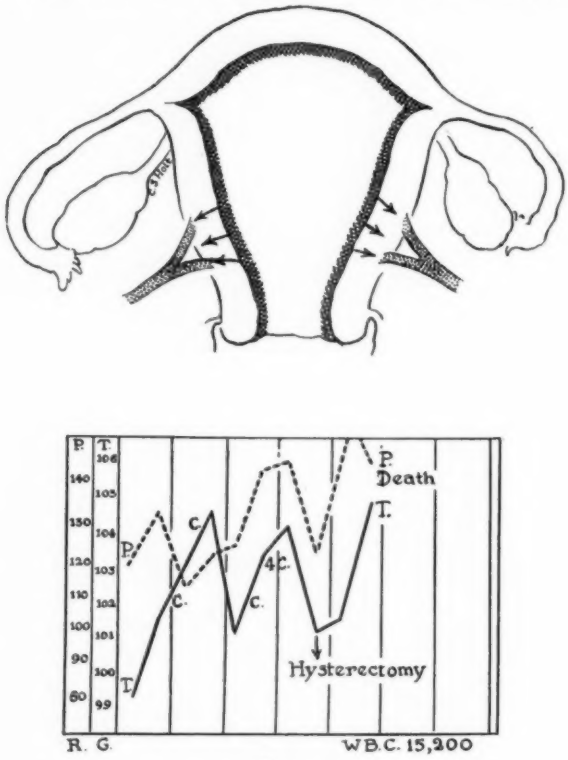


Fig. 7.

This patient died from shock and an acute respiratory complication.

Case x. H. C., forty-three years old, colored, para iii, gravida v, had an eight months pregnancy and rising blood pressure. When the blood pressure reached 200/124, a Voorhees bag was inserted to induce labor. After twenty-six hours the bag was removed and the B.O.W. was ruptured. Small doses of pituitrin and hot soapsuds enema also failed to induce labor. The temperature rose gradually to 103°F. and pulse rate to 120, in spite of 300 c.c. 25 per cent glucose intravenously and hypodermoclysis of 1200 c.c. normal saline and 200 c.c. 5 per cent glucose. Since induction of labor failed and infection was present a supravaginal hysterectomy was done without open-

ing the corpus, with drainage of the abdominal wall only. The uterine cavity contained gaseous, foul, cloudy aminotic fluid and a dead

and pelvic examination found no palpable broad ligament or adnexal masses, it was thought that she had an infection limited to the uterus. The

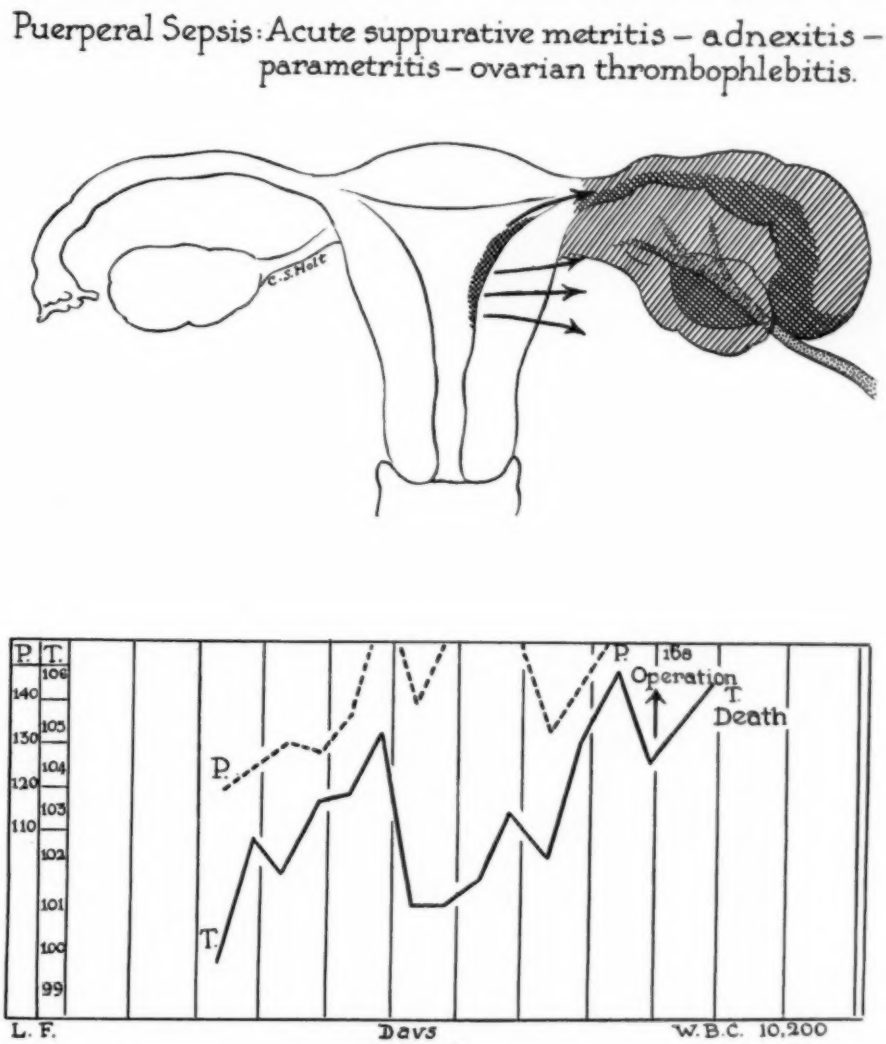


Fig. 8.

macerated baby with bullae. Culture of the uterine lining revealed a hemolytic streptococcus and B. Welchii. Gas bacillus antitoxin, 10 c.c., was given. The patient gradually improved, becoming afebrile on the seventh postoperative day. She developed a wound infection which kept her in the hospital until the thirtieth postoperative day.

Case xi. L. F., twenty-three years old, white, para 1, gravida 11, entered the hospital as a septic incomplete abortion, having had a fetus removed two days previously. A conservative course was followed because of her fever although she continued to pass blood clots, indicating the presence of placental remnants. Since her septic course persisted for eight days

temperature was 101.6°F., pulse rate 168 and white blood count 10,200. Although the prognosis was poor, surgery was considered as a last resort. At operation a suppurative metritis, right suppurative salpingitis and parametritis with a right ovarian thrombophlebitis and retained piece of placental tissue on the right uterine wall was found (Fig. 8). The right ovarian vessels were ligated high i.e., above the thrombosed part of the vein followed by a right salpingo-oophorectomy, subtotal hysterectomy and drainage through the vagina and lower abdominal wound. A blood transfusion was given. There was no improvement in the condition of the patient and she died thirty-three hours later.

It is interesting to note the marked involvement of the uterus and adjacent tissue in the short time following the abortion (nine days) and that surgical intervention was too late. The rapid extension of the infection would indicate that curettage would only have hastened the course, since the infection was in the uterine wall.

Hysterectomy for certain types of puerperal sepsis may be indicated if the diagnosis is made early enough, and not as a last resort procedure. Intramural uterine abscesses and thrombophlebitis of the placental site with infected retained placental tissue are conditions warranting such a procedure. Baldwin reviewed the literature and reported 90 operative cases up to 1929, with a mortality of 25.5 per cent and considered these cases to be all of the fatal type. Gueniot, reported in 1935, a series of 13 patients with only 2 recoveries, having worked on the basis that only when all other treatments have failed and the condition was very serious would he employ surgery. It is, therefore, evident that the diagnosis of the pathology present and of the time to operate is paramount. Although it may again be emphasized that hysterectomy is a serious operation in a septic patient, but under the conditions described it may be necessary.

SUMMARY

1. Surgical treatment should be utilized in certain types of puerperal sepsis in conjunction with medical treatment.

2. These certain types of puerperal sepsis can only be determined by history and careful repeated examinations with thorough and constant clinical study.

3. The operations employed were colpotomy, abdominal drainage and removal of diseased structures and hysterectomy, each operation being employed in certain pathological states. Vein ligation has been done so rarely in this hospital and the mortality in these people so low that the tendency is entirely towards conservative management with repeated blood transfusions.

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CORKSCREW-BOLT FOR COMPRESSION-FIXATION OF FEMORAL NECK FRACTURES

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MUCH evidence supports the contention that compression of fracture surfaces against each other expedites healing. In the treatment of practically all lower extremity fractures, compression in the form of early weight bearing has been a cardinal principle whenever position or immobilization are not jeopardized thereby. Compression has been the purpose of many recent innovations in the treatment of hip fractures and Pawels-Aachen has successfully demonstrated the value of this objective. Converting the fracture line to a horizontal position by osteotomy, he has secured rapid healing in a large proportion of old ununited cases. The Cotton impaction method, the ambulatory spica as used by Bohler and others, may similarly be interpreted as conservative attempts to minimize the shearing action of weight bearing and muscle pull, utilizing this force for compression.

The development of internal fixation instruments, the Smith-Petersen nail and the various wire modifications, has been reviewed in a previous communication. Their many advantages have been offset by the mechanical inability of these devices to adequately utilize the force of compression. Although Smith-Petersen has advised that the flanged nail be inserted low in the trochanter for this purpose, compression cannot be sustained reliably because of the poor friction between smooth metallic surface and trabecular bone. Various types of lag bolts employing a machine screw to anchor the capital fragment have been described by Henry, Jones, and recently by Henderson. But the machine screw has repeatedly demonstrated that it cannot be depended upon to retain a firm grip on cancellous bone over a long period of time.

THE CORKSCREW-BOLT

The instrument used in the cases herewith described was developed to overcome these objections and has undergone only slight modification since its first application in March, 1935, and description in May, 1936. Prior to its first clinical application, cadaver tests were performed and the device was proved practicable.

In brief recapitulation, the head of the instrument consists of a heavy corkscrew of reduced pitch composed of four complete turns (Fig. 1) intended to grasp the capital fragment. The corkscrew is built in ½ and ⅜ inch diameter, the size used depending upon the density of the bone of the patient. This device furnishes the broadest possible surface area at right angles to its axis for maintaining a grip on trabecular bone. It is ideally suited to penetrate into trabecular bone tissue without the necessity of prior drilling and with minimal trauma to the bone. This corkscrew portion is followed by a smooth circular shaft, the posterior portion of which is threaded and subsequently squared so that the thread is preserved on the corners. The squared shaft receives the trochanter catch and the thread, the nut which follows.

The trochanter catch possesses a square hole accurately fitted to the squared shaft permitting only sliding motion. The catch is also equipped with a sharp spur to sink into the bone of the trochanter. It has the double purpose of compressing the trochanter against the capital fragment as well as that of locking the entire instrument so that, when in place, no rotation of the screw can occur. This catch is pressed into position by the nut which articulates with the thread of the shaft. High carbon steel

has been used for these instruments. Experiments are now being carried out with the harder forms of stainless steel.

Fig. 1. Corkscrew-bolt.*

The essential armamentarium for the insertion of the screw is illustrated in Figure 2 and requires little description. The hand wrench for insertion of the screw has a particularly long chuck and may be released from the screw with the hands of the operator remote from the wound. The top wrench employed for tightening the nut possesses a hollow shaft to receive the shank of the screw. A plier has been useful for inserting and removing the trochanter catch, and an end cutting pliers easily nips off excess shaft when insertion has been completed.

Application. The "closed" method of insertion is favored by the author and has been employed in all cases. This technique, described in a previous communication, is in no way original. For insertion, the instrument is disarticulated and the screw portion clamped in the hand wrench. A small drill or punch perforation in the femoral cortex suffices to introduce the instrument into the bone, from which point its course may be directed by *x*-ray or fluoroscope. Control of depth is easily followed on the roentgenogram and calibrated by estimating the number of turns required. When the shaft is in position, the trochanter catch is slid upon it. The nut, which is then screwed down tightly into position with the top wrench, squeezes the fracture surfaces against each other.

CASE REPORTS

Seven fresh intracapsular fractures were treated with this instrument during the year, from March, 1935 to March, 1936. In these early experimental cases continuous surveillance was considered important and there was consequently no curtailment of the customary long period of hospitalization. Moreover, the security of the fixation produced by this instrument being untried, supplementary fixation was employed in the early cases. A low plaster spica was first used; then Buck's extension was substituted. When this proved unnecessary it was also discarded so that in the last 2 cases no external fixation was used.

One case, treated by another surgeon, was omitted from the following series because the instrument failed to penetrate the capital fragment which consequently was not immobilized, and non-union resulted. The remaining 6 cases constitute the basis of the following report.

Case 1. R. D'A. male* (Fig. 3), forty-six years of age, entered the hospital March 2, 1935, immediately after an automobile accident because of pain in the left hip. Roentgenograms revealed an intracapsular fracture of the left hip. On the following day with the patient under avertin anesthesia on the fluoroscopic table the fracture (Fig. 3A) was reduced by the Leadbetter technique, *x*-ray pictures confirming complete reduction. A 2-inch lateral subtrochanteric incision was then made and, under fluoroscopic control, the described instrument was inserted. The wound was closed with fascial and skin sutures and subsequent roentgenograms showed that complete reduction had been maintained and that compression of about ¼ inch had been obtained (Fig. 3B). The operation including time for developing the *x*-ray films, occupied less than an hour. Convalescence was uneventful except for a slight serous discharge which persisted for about five days. Two and one-half weeks after operation an *x*-ray picture disclosed that the device had bent slightly at the junction of the corkscrew and the shaft, due to a defect in the instrument which has since been remedied. For this reason a short spica plaster was applied to give security. Three months after operation *x*-ray examination (Fig. 3C) disclosed firm healing and the plaster spica was removed. At four months walking with crutches was permitted and were discarded after two weeks.

Examination six months after operation revealed three-quarters normal motion, the

* Manufactured by Geo. Tiemann & Co., New York.

* Previously reported

patient walking without a limp and complaining only of occasional pain where the tail of the screw abutted against soft tissue.

plete restituto ad intergram. *X*-ray pictures showed complete healing and no deformity of the capital fragment. Motion was normal but

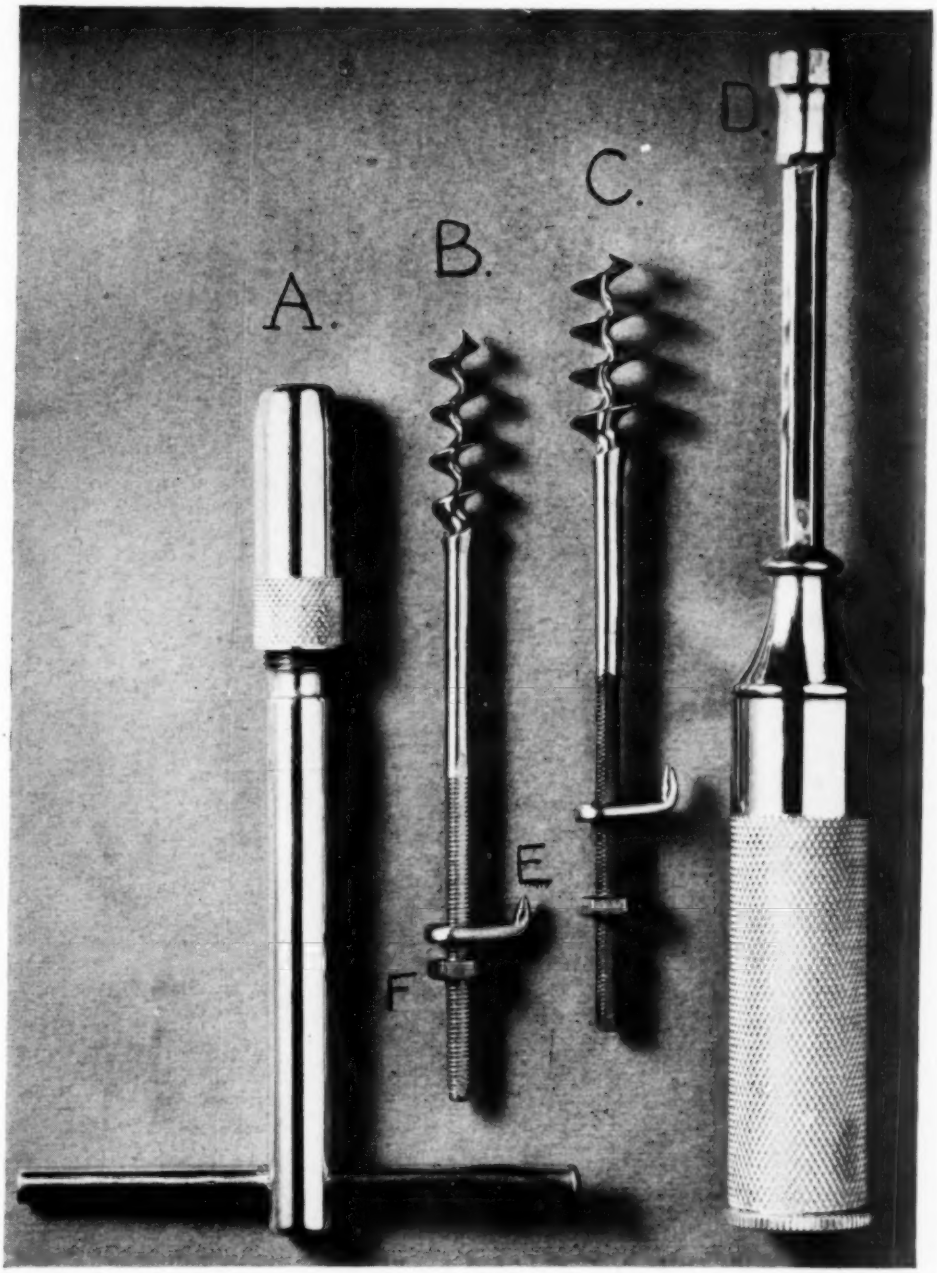

Fig. 2. Armamentarium; A, hand wrench; B, 3/8 inch diameter corkscrew pin; C, 1/2 inch diameter corkscrew pin; D, top wrench for turning nut; E, trochanter catch; F, nut. (Lippmann, R. W. *Jour. Mt. Sinai Hosp.*, 3: 65 [July-Aug.] 1936.)

Because of persistence of this pain, at eleven months after operation the bolt was removed. No difficulty was encountered though the instrument was tightly held by the bones.

When observed sixteen months after operation this patient gave every evidence of complete restitution ad integrum. Roentgenograms showed complete healing and no deformity of the capital fragment. Motion was normal but for slight limitation to full internal rotation. He had no further complaint referable to his injury and has been driving a taxicab for four months.

Case II. J. V. female, aged seventy-six years, was seen in March, 1935, because of pain

in the left hip and inability to stand following a fall two hours before. Clinical signs were typical of a hip fracture and the diagnosis was at this time, evidence of severe infection was lacking. The patient immediately complained of increased hip pain and the leg shortened and

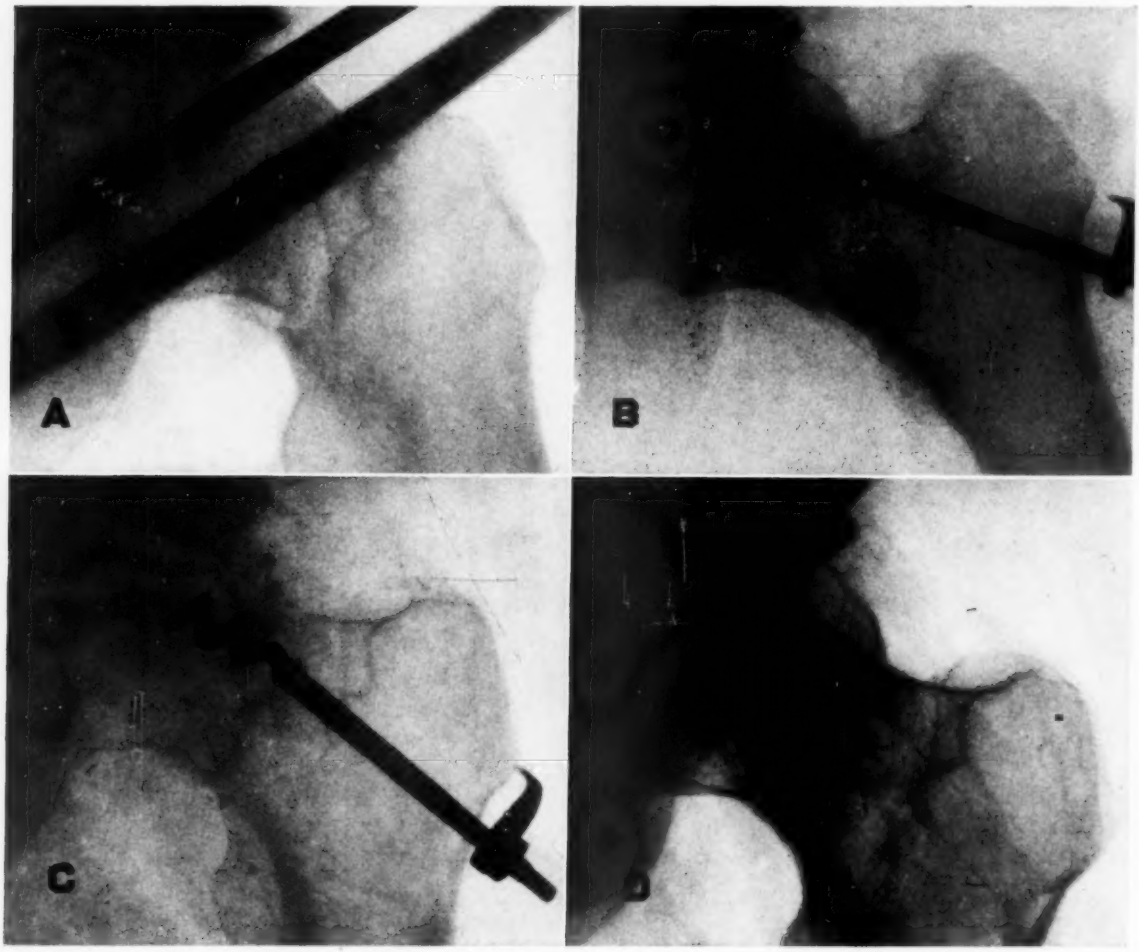

Fig. 3. Case I. A, Original fracture; B, reduction with screw in position; C, three months later; D, eleven months after injury, the bolt has just been removed.

confirmed by *x*-ray pictures, which suggested impaction, but clinically there was shortening, marked eversion and inability to lift the leg. Preceding the accident was a vague history of illness and weakness, though on examination, a temperature of over 101 degrees and a hemoglobin of 30 per cent were the only unusual findings. Internal fixation was performed on March 26, attaining excellent position and fixation but the postoperative course became immediately unsatisfactory. On the fifth day the wound was opened and about 5 c.c. of seropurulent material was evacuated. In spite of energetic treatment including multiple transfusions and Carrel-Dakin irrigations, progress was unfavorable. Twelve days after operation, the pin was removed, though even rolled into eversion. Coma and death ensued three weeks after injury, probably due to inability to resist a comparatively mild infection.

Case III. I. W., male aged fifty-six years, twelve hours before admission on November 17, 1935, was swinging an iron bar in a cellar and slipped on the wet concrete floor. He was unable to rise unassisted. The hip pain grew more severe until admission to the hospital was sought. On examination the patient presented one inch shortening, inability to lift the leg, marked eversion and severe tenderness about the hip. In addition, severe chronic bronchitis and hypertension were noted. *X*-ray pictures showed intracapsular fracture of the right hip. (Fig. 4A.)

On November 20, operation was performed by the described technique and followed by Buck's extension (6 lbs.) to the right leg (Fig. 4B and C). Aside from a temperature rise to 101.4 degrees on the second day after operation, the postoperative course was entirely uneventful. The traction was gradually diminished to three pounds after one month, and after six weeks the patient was permitted to sit in a chair. Weight bearing was not permitted until February 20, three months after operation.

Examination on April 24, five months after operation, revealed normal gait, a point of tenderness where the end of the pin could be palpated under the skin, and some limitation to internal rotation of the leg, but no pain. X-ray studies at this time still revealed a thin line of diminished density at the fracture site (Fig. 4D).

This patient was last examined ten months after operation: there were no complaints, the hip joint was insensitive and motion was full, except for slight limitation of internal rotation. The patient was again engaged in his former occupation.

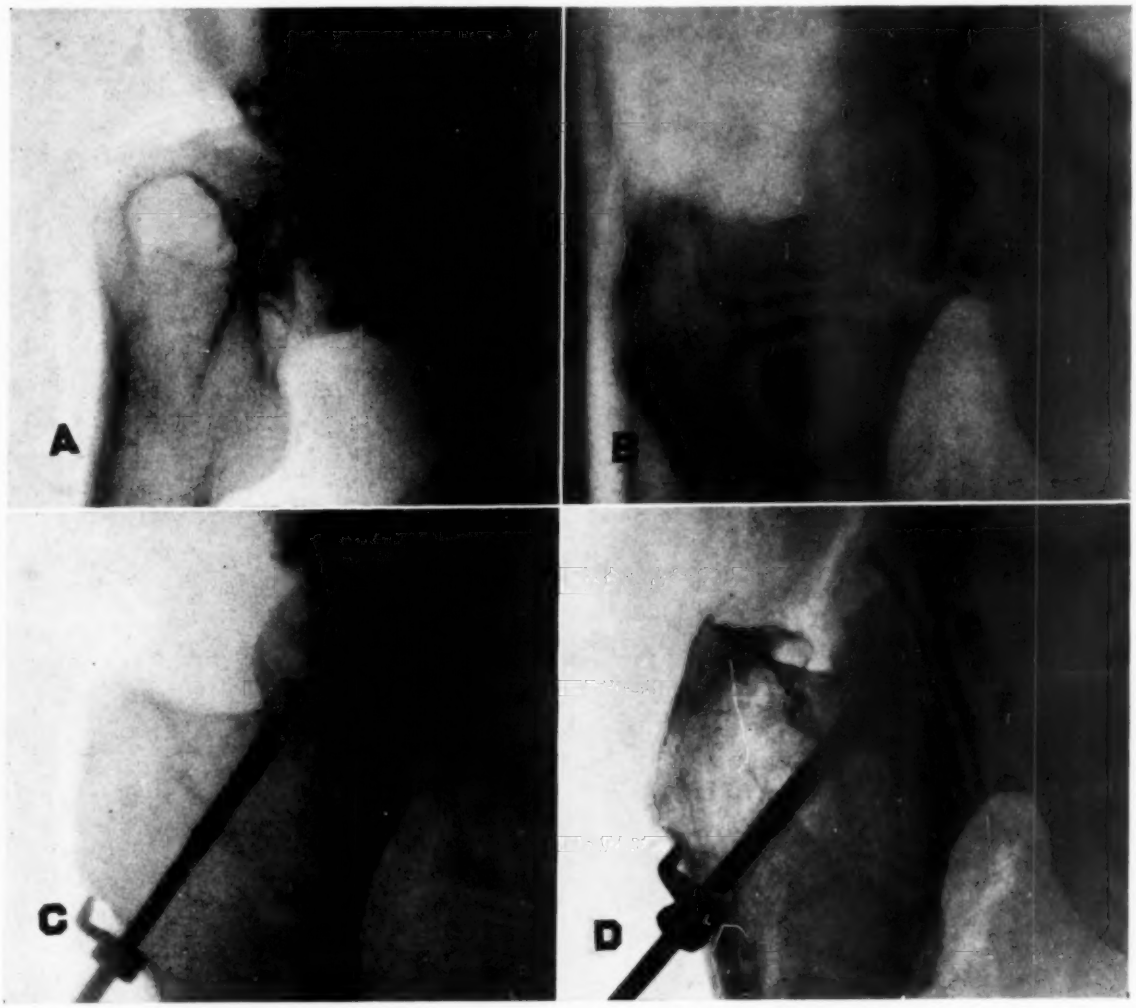

Fig. 4. Case III. A, Original fracture; B, reduction; C, corkscrew-bolt in position; D, five months after injury.

Case IV. M. L., female, aged fifty-two years, sustained an injury to the left hip in a fall out of bed twelve hours before admission on December 5, 1935. Examination showed the usual findings, eversion, inability to left the leg and ½ inch shortening. Roentgenograms disclosed a high intracapsular fracture (Fig. 5A). Operation as described, was performed on the following day, with excellent reduction and immobilization (Fig. 5B). No postoperative immobilization was employed and the postoperative course was entirely satisfactory, the temperature remaining below 100 degrees.

Through error, however, a highly tempered experimental screw had been misplaced in the operating set and employed.

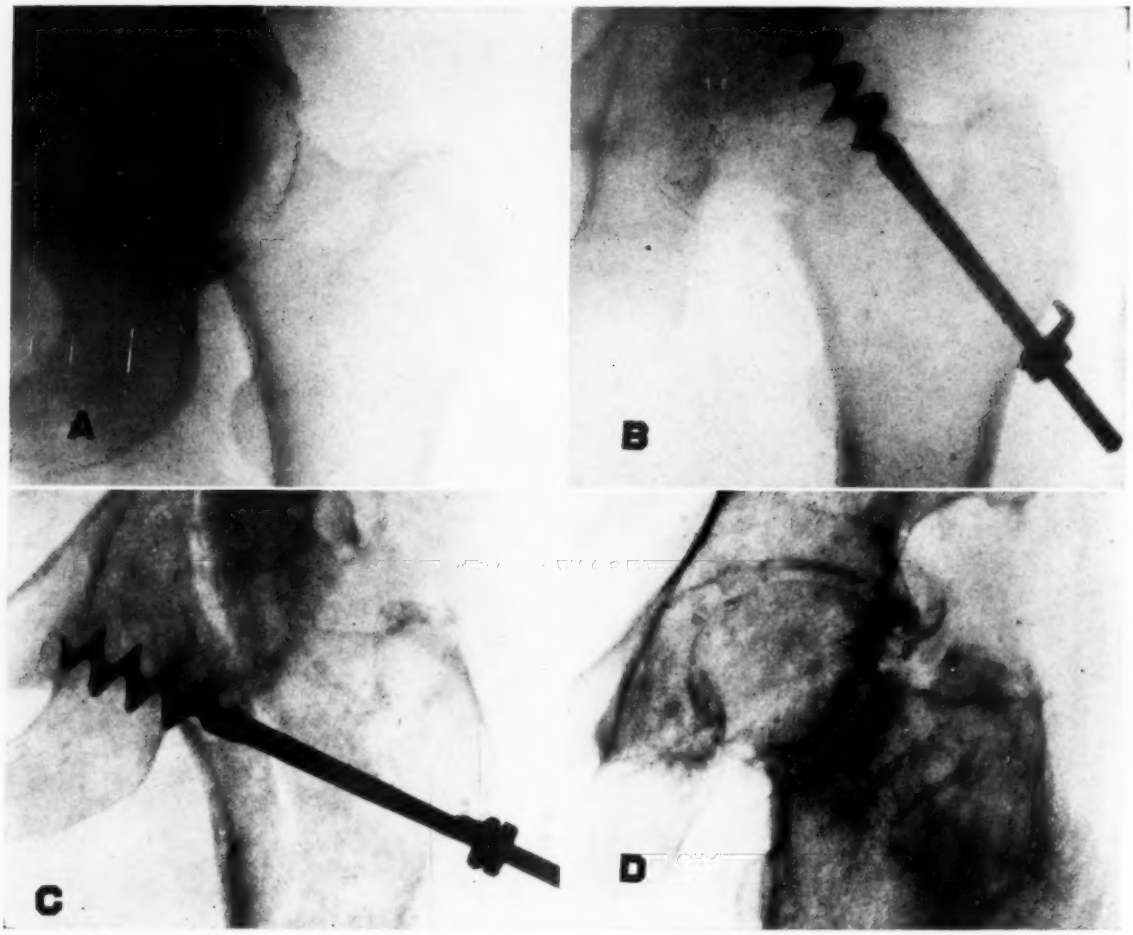

FIG. 5. Case IV. A, Original fracture; B, reduction and compression-immobilization; C, re-application after fracture of tempered instrument; D, questionable union seven months after injury.

Three weeks after operation, the hip suddenly became painful and eversion and shortening manifested themselves. *X*-ray pictures disclosed that the screw had broken at the fracture site. On December 28, through a Smith-Petersen incision, the fragments were removed with some damage to the cancellous bone of the femoral head. For this reason, insertion of another pin was performed with the corkscrew portion in the acetabulum, rather than the femoral head. Considering this immobilization undependable, a low plaster spica was added for re-inforcement. The spica remained in place for three months and was then removed. X-ray pictures showed incomplete healing (Fig. 5C) and the screw was permitted to remain in place for another month. On its removal, four months after insertion, there was every clinical evidence of union, though the *x*-ray pictures were equivocal. The hip and knee were extremely stiff as the result of four months of complete immobilization, and active physiotherapy was instituted and walking commenced. Steady but slow progress was manifested and at the last examination, July 30, 1936, seven months after operation, the patient still complained of stiffness and pain in the hip and knee. She walked with a slight limp favoring the left leg. Actual shortening measured one-half inch. There was no tendency for the leg to evert. Flexion at the hip was limited by pain at 30 degrees. Rotation was about one-third normal, as was adduction and abduction. Flexion at the knee was limited at 110 degrees. *X*-ray pictures at this time still showed the fracture line, but there was no change in the relative position of the fragments since removal of the pin three months before (Fig. 5D). While

clinically, union appeared firm, the roentgenograms render it necessary to defer decision regarding the presence of bony union. Active physiotherapy was continued.

assistance. On postoperative *x*-ray studies, the reduction appeared satisfactory on the anteroposterior aspect (Fig. 6B). Lateral views, (Fig. 6C), however, showed an angle of 70

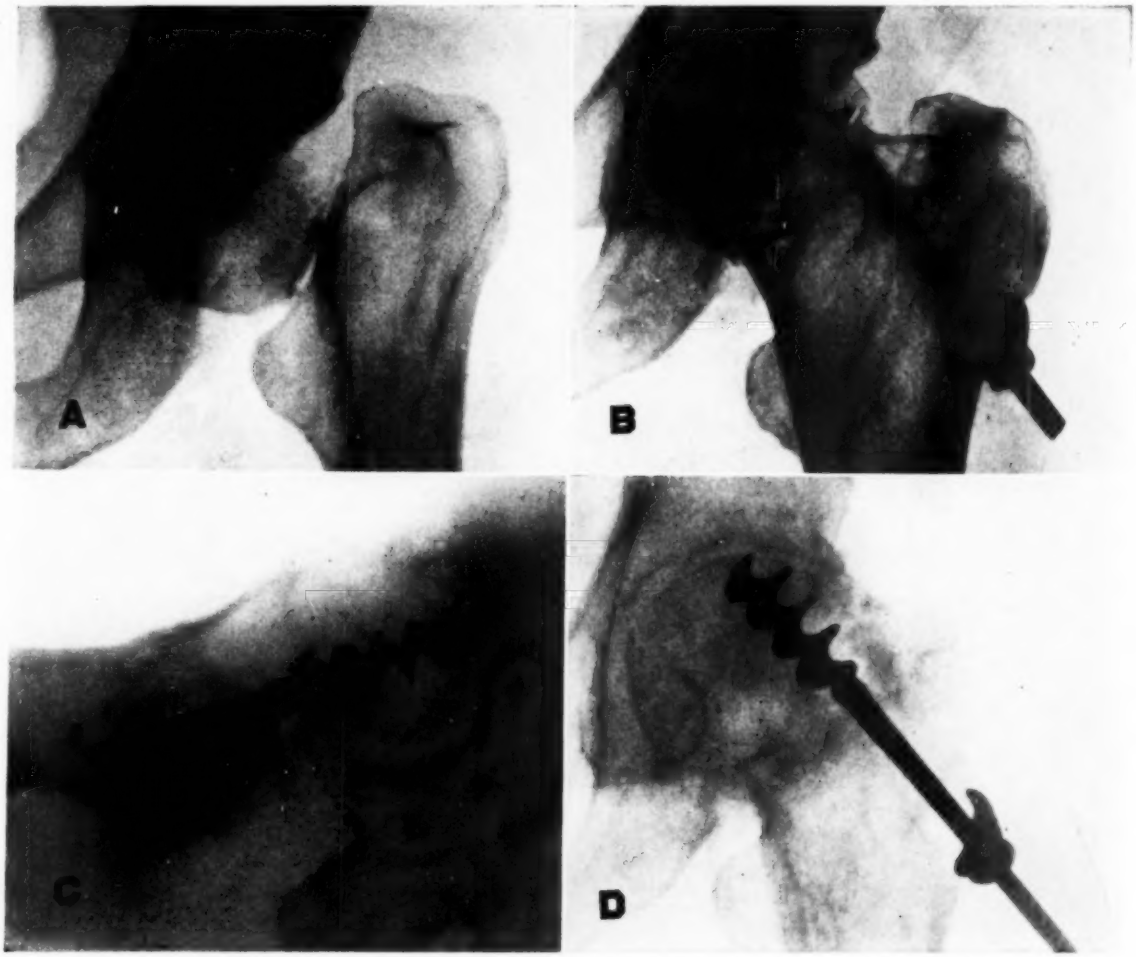

Fig. 6. Case v. A, Original fracture; B, reduction and compression with corkscrew-bolt in position; C, latera view showing very poor reduction but solid immobilization; D, healing five months after injury.

Case v. B. N., female aged seventy-three years, was knocked over by an iron gate on January 22, sustaining a fracture of the right hip. Inability to use the leg for two days thereafter, caused her to seek medical attention and she was referred to the hospital on January 24, presenting the symptoms and signs characteristic of hip fracture. There was 1½ inches shortening and roentgenograms showed intracapsular fracture with severe displacement (Fig. 6A). Preceding injury the patient had manifested marked senility changes and severe hypertension. Internal fixation was decided upon by the attending surgeon and was duly performed on January 24. Reduction was followed by insertion of the screw with my

degrees formed by the fragments, the distal one remaining everted. In addition, the tip of the corkscrew appeared almost to impinge upon the hip joint. Nevertheless, the fragments were well held together by the instrument and further manipulation was deemed inadvisable. The postoperative course was complicated only by the patient's mental condition, i.e., inability to assist herself in any degree, even sitting-up in bed requiring the assistance of several nurses.

On February 29, the pin was withdrawn a short distance, to avoid proximity to the joint surface. No external immobilization was employed, the hip being painless, and there were no postoperative complications.

Cooperation of the patient improved somewhat as time progressed and she was able to get into a chair two months after the original operation. Roentgenograms indicated that union was progressing in spite of the poor position.

On June 18 the fracture appeared to be solidly healed. Because of her mental status the patient is being cared for in a nursing home. She was seen there a few weeks ago, eight months after injury, by her attending physician, who reported that the hip was entirely asymptomatic. A record of hip motion in degrees was not obtained but motion appeared to be satisfactory and close to normal. The patient walked with minimal limp and was able to care for herself entirely without hip discomfort. Eversion of the leg persisted which undoubtedly resulted from incomplete reduction.

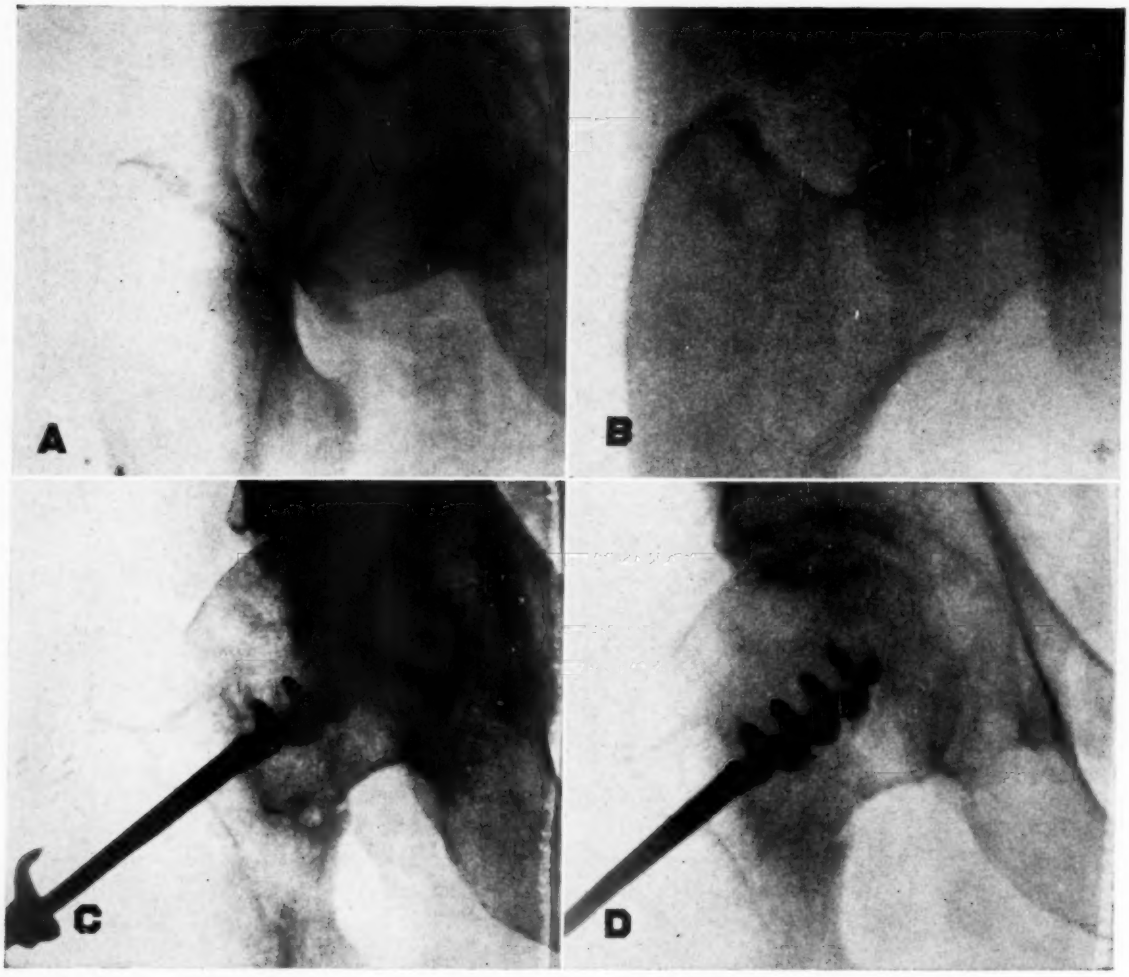

Fig. 7. Case vi. A, Original fracture; B, reduction; C, six weeks later beginning union; D, five months after injury; hip asymptomatic.

Case vi. H. R., male, aged seventy-nine years, sustained a fracture of the right hip following a fall on a curbstone on February 12, 1936. He was unable to rise without assistance and several hours later when seen he presented the signs and symptoms typical of hip fracture with marked shortening. Roentgenograms showed intracapsular fracture with considerable displacement of the fragments (Fig. 7A).

Internal fixation by the described technique was performed several hours later (Fig. 7B). The postoperative course was complicated for several days by auricular fibrillation which, however, responded promptly to digitalis therapy. No external immobilization was employed, the patient sitting up on the day following operation. He was kept in bed for four weeks and then permitted to walk with

crutches. Two weeks later the crutches were discarded.

Examination six weeks after injury revealed no limitation of function, no shortening, and slight limp favoring the right leg. Roentgenograms at this time showed only a slight diminution of density at the fracture line (Fig. 7C).

Ten weeks after injury, the only manifestation of pre-existing fracture was the scar of the operative incision.

The last examination of this patient was four months after injury. There was no shortening and motion of the hip was full. *X*-ray pictures showed healing to be complete (Fig. 7D).

A recent communication from this patient, eight months after injury, indicates that he has continued to walk normally. He is unable to note any difference between hip function on either side.*

COMMENT

While these cases are insufficient in number to serve as a basis for statistical study, they do appear to justify certain observations with regard to the instrument. These cases indicate that accurate insertion may be easily accomplished, that, under these conditions, the fracture surfaces are drawn together and impacted, and that this impaction is maintained with no tendency for the instrument to become loose or otherwise displaced after insertion. Moreover, it appears justifiable to conclude that the use of postoperative supplementary fixation is unnecessary.

The impaction produced by the instrument can so obscure the fracture line that it is often difficult to ascertain the time accurately when bony union occurs. The normal gait of patient in Case VI six weeks after injury and the *x*-ray appearance at that time, indicate that the security of fixation may be as dependable as bony union, long before such union can reasonably be expected. If such experiences become sufficiently frequent, the actual time of the occurrence of union may become relatively unimportant.

Moreover, Case V, illustrates that the fixation may be firm enough to overcome the handicap of poor reduction, yielding solid bony union, where such a result could hardly have been anticipated with other methods of immobilization.

Further clinical experiences with the method are essential to determine how early ambulation and weight bearing may be safely permitted.

CONCLUSIONS

Six cases of intracapsular fracture, all treated more than seven months ago, with the corkscrew-bolt are herewith reported. Although insufficient for statistical study the cases illustrate the ease of insertion, the security of the fixation and the lack of need of supplementary postoperative fixation. These observations justify further use of the device.

*Cases I, III, V and VI have been re-examined and *x*-rayed since submitting this paper for publication. Union had remained firm in all instances. The bolt which was not removed from patients in Cases V and VI had remained in situ for fifteen months in patient in Case V and thirteen months in the patient in Case VI. In neither instance was atrophy or any alteration in position of the device perceptible, nor had it caused any discomfort.

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AMBULATORY TREATMENT FOR ANAL FISSURE

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THE treatment of fissure in ano has usually been one of operation or palliation. The purpose of this paper is to instill a greater interest in a method of ambulatory therapy.

Anal fissure is a common condition to which the human being is susceptible. It is not rare to find several members of the same family afflicted with this minor but annoying ailment. The pain and discomfort are intense and are due to the exposure of the nerve fibrils in the open wound. The pain is very often brought on with the commencement of the act of defecation and remains at its termination, lasting for an hour or even longer. With the initiation of this subjective sensation, the sphincter is thrown into spasm and thus interferes with the healing process of the ulcer at the anal orifice.

The patient suffering with a fissure in ano is invariably constipated. Not alone that, but he develops a fear of bowel movements and, therefore, restrains himself in order to avoid the discomfort that is associated with defecation. Thus, a vicious cycle is established wherein constipation becomes persistent.

The usual site, though not the only one, for the fissure is at the posterior commissure. The fissure is triangular with the apex pointing inward from the anal orifice and is about 1 to 2 cm. long. A fissure is often protected and hidden by a so-called "watchman," which is a projection of skin in front of it, known as a sentinel pile.

The basis of all forms of therapy is to rest the sphincter in order to permit the process of repair to progress unimpeded. One may excise the fissure bed or curette it; or leave it alone and instead stretch the sphincter or even cut the external fibers thus paralyzing it temporarily and obtaining the necessary rest for healing.

However, many individuals are reticent about submitting to an operation or to anything that sounds like one for the relief of this condition. Again, many others cannot find the opportunity or the time to have themselves attended to. Rather than the above, they rely on the use of various combinations of drugs, either in suppository or ointment form, which prove to be only temporizing measures. Patients are more apt to allow themselves to be treated, therefore, if they are told that by means of injections, which will not incapacitate them, alleviation of anal fissure can be accomplished.

The method is simple and entails the use of a solution of an anesthetic drug in an oily base, which will cause a slow absorption of the substance. This will extend the period of anesthesia and ease the spasm of the anal sphicter over a sufficient time to allow healing.

In 1935 Steinberg[1] reported the treatment of a series of 60 cases with various solutions; 30 of the patients being treated with a solution of nupercaine in oil, and 30 with a solution containing benacol. He compares the immediate discomfort attending the use of both materials, stressing the favorability of the former. Frankfeldt[2] in 1934, cited the use of Gabriel's solution in cases of anal fissure and ulcer and in rectal neuroses. I have used two preparations, both of which have shown themselves to be capable of producing a local anesthetic effect lasting for a long period of time; one injection causing a cessation of pain and

[1] STEINBERG, N. The use of Gabriel's modified solution in the treatment of fissure in ano. *New England Jour. Med.*, 213: 162–164 (July 25) 1935.

[2] FRANKFELDT, F. M. The use of Gabriel's formula in proctology. *Internat. Jour. Med. and Surg.*, 47: 213 (April) 1934.

spasm often for as long as four weeks. The solutions employed are:

I. Nupercaine, base..... 0.5 per cent
Phenol..... 1.0 per cent
Benzyl alcohol..... 10.0 per cent
Oil of sweet almond... q.s.
Nupercaine is *a*-butyloxycinchoninic acid diethyl-ethylenediamide.

II. Eucupin, base..... 0.2 per cent
Ethylaminobenzoate... 3.0 per cent
Benzyl alcohol..... 5.0 per cent
Oil of sweet almond... q.s.
Eucupin is isoamylhydrocupreine, an alkaloid synthetized from quinine.

PROCEDURE

A few words will suffice to describe the procedure. Assuming that the fissure is situated at the posterior commissure, the patient is placed either in the lithotomy or Sims' position. A 1½ inch, 22 gauge needle is inserted through the normal skin (Fig. 1) about 0.5 to 1 cm. distal to the fissure edge, and 1 minim of the solution is injected immediately. This will allow the introduction of the left index finger through the anal orifice and thus enable one to feel the needle, guiding it as it is advanced in the subcutaneous tissue to a place just beyond the apex of the fissure, but at no time to penetrate the fissure bed or mucous membrane. One-half to one cubic centimeter of the selected solution, depending on the size

TABLE I

THIRTY CASES TREATED WITH THE NUMBER OF INJECTIONS RANGING FROM TWO TO FIVE, AND THE PERIOD FOR REPAIR FROM THREE TO ELEVEN WEEKS, AND WITH TWO COMPLICATIONS

Case	Solution Used	Duration of Symptoms	Number of Injections	Total Amount of Solution in C.c.	Healing Time in Weeks	Complications Before or as a Result of Treatments	Follow-up 6 Months After
1. S. W....	I	4 years	3	15	6	0	No recurrence
2. J. M....	I	2 years	3	12	5	0	No recurrence
3. M. P....	I	2 years	3	9	4	Sentinel pile	No recurrence
4. M. K....	I	4 years	4	20	10	*Slough and fistula	Slight itching
5. J. L.....	I	1 year	2	8	4	0	No recurrence
6. H. H....	I	8 months	2	6	4	0	No recurrence
7. E. K....	I	1 year	2	9	5	0	No recurrence
8. M. B....	I	1 year	2	6	3	0	No recurrence
9. S. F.....	I	4 years	4	12	6	Sentinel pile	No recurrence
10. H. N....	I	5 months	2	6	4	0	No recurrence
11. R. S....	I	3 years	4	12	5	Pruritis ani	No recurrence; pruritis same
12. R. H....	I	2½ years	5	15	8	Sentinel pile	No recurrence
13. S. P.....	I	4 months	2	6	3	0	No recurrence
14. H. R....	I	7 years	5	15	7	Sentinel pile	Occasional itching
15. R. P....	I	6 months	2	6	3	0	No recurrence
16. B. S....	I	8 months	3	9	4	0	No recurrence
17. F. K....	I	1 year	3	10	6	0	No recurrence
18. C. B....	I	3 years	5	14	9	0	Occasional itching
19. S. G....	I	2 years	3	9	5	Sentinel pile	No recurrence
20. L. M....	I	14 months	3	9	4	0	No recurrence
21. M. F....	I	1 year	3	9	6	0	No recurrence
22. R. M...	II	2¼ years	5	20	11	*Slight slough	No recurrence
23. H. C....	II	4 months	2	6	4	0	Slight itching
24. N. F....	II	7 months	2	6	4	0	No recurrence
25. E. S....	II	3 months	2	6	4	0	No recurrence
26. J. H....	II	1½ years	4	12	6	0	No recurrence
27. A. O....	II	14 months	3	9	5	0	No recurrence
28. M. F....	II	9 months	2	6	4	0	No recurrence
29. P. B....	II	2 years	4	12	6	Sentinel pile	No recurrence
30. L. F....	II	6 months	2	6	4	0	No recurrence

* Complication developing as a result of treatment.

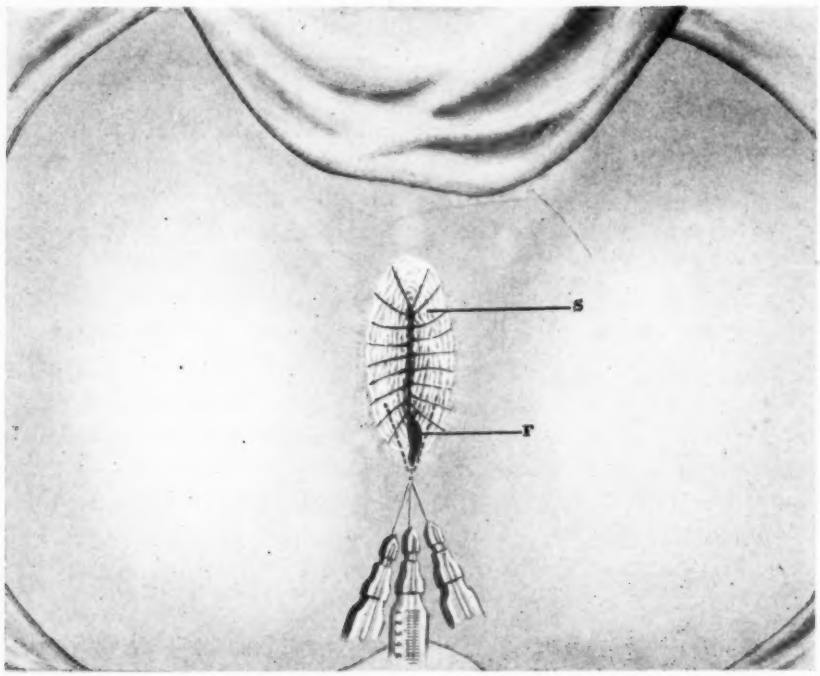

FIG. 1. Showing the various positions of the needle injecting beneath the fissure and the sphincter on either side of the fissure. S, sphincter muscle; F, fissure.

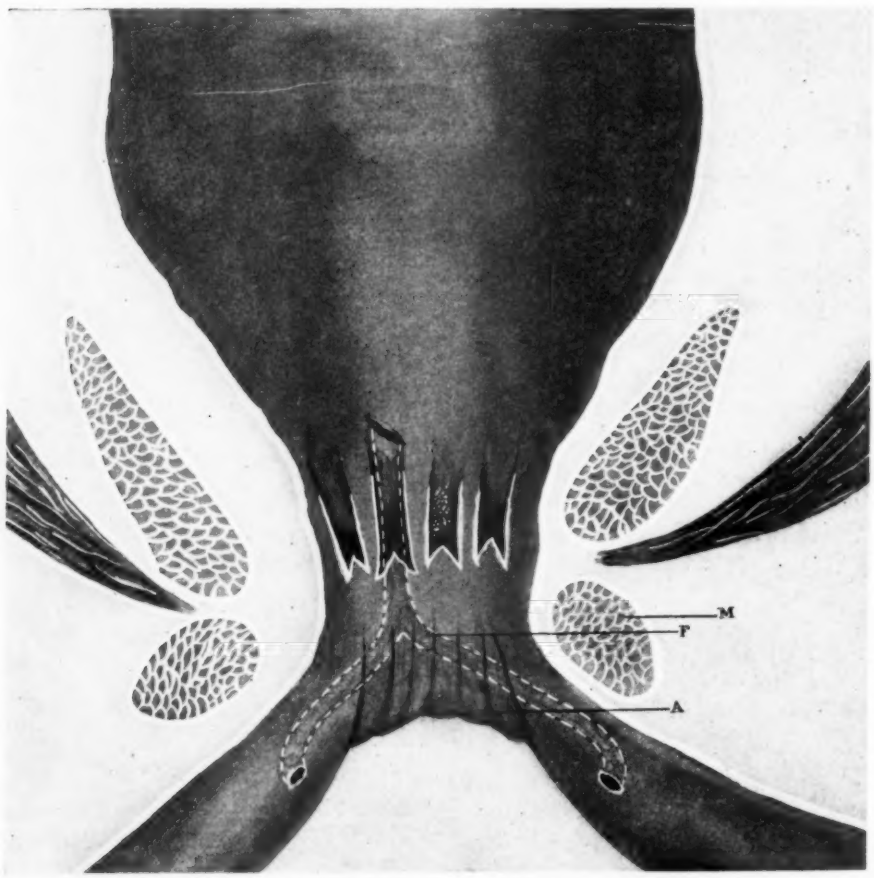

FIG. 2. Showing the fistulous tracts in the shape of an inverted "Y." A, anus; F, fistula; M, muscle.

of the lesion, is then injected beginning at the point beneath the fissure apex and continuing as the needle is being drawn out. The needle is then partially withdrawn, and with the index finger still in the anus, it is redirected, guiding it into the sphincter muscle, first on one side adjoining the fissure and depositing 1 c.c., and then on the other side adjoining the fissure. Too much solution should not be injected; nor should the solution be placed intracutaneous or superficial because it will result in sloughing. It is far better to use smaller quantities and repeat the injections in seven to ten days. It may take from one to four or five treatments until complete repair of the fissure is accomplished. The important fact is that following the first treatment the patient has immediate relief, gains confidence, and bowel movements are characterized by the absence of ensuing pain, smarting or soreness.

If a sentinel pile is present, its removal is essential for cure. This may be done after the first injection or at a date in the near future; no additional infiltration of a local anesthetic is required for its ablation.

In the series of 30 cases treated with these formulas, there were 2 cases which developed sloughs (Table 1). This complication prolonged the period necessary for repair but the terminal result was good. Three of the patients seen after six months complained of an occasional itching sensation which was slight and not constant, examination revealed no break in the mucous membrane; another said she had itching only when she permitted herself to become constipated, however here, too, there was no recurrence of the fissure. One of the first cases (Table 1, Case IV) was an anal fissure of four years duration situated at the posterior commissure and with a large sentinel pile. This patient received 5 c.c. injections; the sentinel pile was removed after the second treatment. After the fourth injection, a slough was noted, followed by the development of a fistulous tract, having the shape of an inverted "Y." Undoubtedly, this was caused by the large amount of material used at each sitting. Without any additional anesthetic, the tracts were laid bare (Fig. 2). From then on, healing took place without any further difficulty.

CONCLUSION

I believe that the treatment of fissure in ano is feasible by injections of local anesthetics in oil, and can be accomplished in the ambulatory patient. This method is not complicated, and appeals to the patient because it is non-incapacitating and, therefore, economical. Furthermore, it is important to remember that constipation and anal fissure are complementary, that is, synergistic etiological and maintaining factors for each other and both conditions must be treated at the same time. Finally, large doses produce sloughs, and therefore, it is advisable to use smaller quantities which can be repeated until the ultimate repair of the anal fissure.

TECHNIQUE OF SKIN GRAFTING*

AUBRE DE L. MAYNARD, M.D.

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WITHIN recent years the high incidence of cases presenting large epithelial defects as a result of destructive trauma has served to focus attention upon skin grafting as an operative method that is invaluable in the ultimate solution of these injuries. In the transplantation of skin success should not be a fortuitous occurrence since it can be definitely assured by an understanding of the fundamental principles involved, an exact technical knowledge and care in execution.

In extensive superficial wounds which result from burns or avulsions early closure is imperative. For, as long as the wound exists the patient is menaced by infection, constitutional debility incident to the profound secondary anemia which may develop, and scarring. Consequently, when skin is transplanted, it is highly necessary that there should be "takes" and not "failures."

While discussing basic principles I purpose to set forth simple and sound procedure, which has been consistently successful during the past six years.

When skin is applied to a wound surface it will either live or die; if it lives, we ordinarily refer to it as a "take" which means that the transplant, having been successfully vascularized from the wound or receptor site, is enabled thereby to live. The important purpose of the transplant after a "take" is, necessarily, growth. This takes place by amoeboid movement into the wound, of cells from the periphery of the epithelial graft. These moving cells in association form an extension membrane. According to Hartwell,[1] the process of covering with epithelium is completed by the union of two or more such membranes from opposite sides of the wound, followed by the rearrangement and multiplication of cells of the membrane.

There are three important factors which determine a "take": (1) vascular "succulence" or capillary richness at the receptor site; (2) infection; and (3) technical considerations.

VASCULAR "SUCCULENCE"

To obtain a clear grasp of this factor, the pathology of the wound site is considered first. The deposition of the fibrous "fill" or healing cicatrix in a wound in which the epithelial edges are widely separated is through the medium of a vascular tissue rich in fibroblasts or wound macrophages, and with varying degrees of cellular infiltration. Time, and to a limited degree, infection, determine the depth and density of this scar tissue. As this tissue contracts numerous capillaries are compressed in its stratum and the superficial granulations, losing a measure of their pinkness, become rather pallid and do not bleed readily.

The bearing of this wound pathology on skin grafting was first stressed by Bancroft and Rogers.[2] They found that frequently a curet removed only superficial granulations, leaving a scar tissue bed of poor vascularity, obviously an unsatisfactory and undesirable bed for transplanted epithelium. From the study of various wound "fills" (Figs. 1 and 2) one may readily confirm their observations and conclusions.

Under the scar but over the normal tissue, whether fat or fascia, is a stratum of much vascular "succulence." Here the

* From the Surgical Service of the Harlem Hospital, New York, Dr. Charles S B. Cassassa, Surgical Director. Presented before the Post-Graduate Surgical Class of New York University Medical College, Dr. Arthur M. Wright, Professor of Surgery.

capillaries are numerous, and the fibrous content little. Likewise, the wound fibroblasts are more abundant, and cellular infiltration is moderate, with a more copious tissue fluid. In the surgical excision of the scar bed the scalpel is carried down to the capillary stratum which provides an excellent plane of cleavage, the overlying tissue peeling away with ease under sharp dissection. The capillary bed appears grossly as a sheet of pinpoint, multitudinous geysers spouting a continuous flow of blood. Hemostasis is effected by pressure with gauze pads wrung dry from hot physiological salt solution.

Depending upon the extent of wound surface and the coagulability of blood, the fluid loss may be considerable, sufficient, in fact, to produce surgical shock. This occurred in the case of a child of four years of age, with a burn of 10 per cent of the body surface, on the twenty-third day of hospitalization. After the excision of granulations of the hand and forearm in the preparation of the receptor site, the capillary ooze became intractable and failed to yield to the customary pressure tamponade. The patient went into shock and died within ten minutes despite all measures. In 2 other cases shock supervened following blood loss but without fatality.

These 3 cases were typical, all severe burns, hospitalized for over three weeks, showing debilitation and a pronounced secondary anemia. Obviously, such patients as these, because of the morbid factors present, have only a small margin of safety and require judicious surgical management.

After the removal of the fibrous "fills," with hemostasis effected, the surface appears as a dry, fibrin coated, fairly even, approximately smooth and seemingly transparent membrane, the optimum terrain for transplanted epithelium.

From what has been stated, it is not to be construed that grafts will of necessity die on a bed which has not been prepared in this manner. Emphasis is placed upon the procedure because it takes note of the pathology of the wound site and in so doing offers the best vascular bed for the reception of transplants.

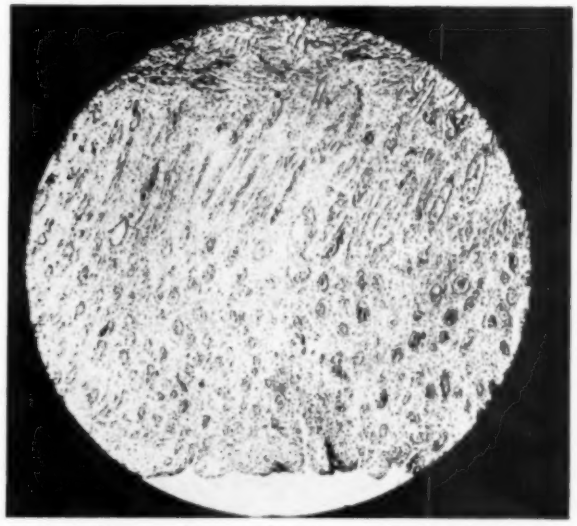


Fig. 1. Photomicrograph of wound "fill" showing dense scar tissue, almost avascular, and subjacent rich capillary bed; the optimum bed for grafts.

INFECTION

Every wound site that requires grafting, presents infection of some degree. When the infection is great, the wound is bathed in pus, detritus and cellular debris, and the wound reaction is distinctly acid. Granulations are obscure, the few that exist being pale and inactive. The epithelial border of the wound may show an inflammatory flush. However, when, through the judicious use of antiseptics on the affected area the infection has been reduced, the wound exudate diminishes, the bacterial and cellular content becomes less, and granulations tend to be more obvious, abundant, active, and of a healthy pink color. Moreover, acidity disappears.

In the preparation of the wound site for grafting, efforts should be directed toward the reduction of infection to a minimum. It is current opinion that the bacterial count need not be considered the criterion in determining whether or not a wound is ready for grafting.[3] From a practical point of view, it is far better to treat a wound until it shows a clean and healthy appear-

ance, one in which the exudate is slight, the granulations pink or red and compact, and the skin bordering the wound free from inflammatory flush. It is extremely desirable that the wound reaction approach that of normal tissue fluid. This, however, is generally the case.

All incidental technical factors having been controlled, it must be borne in mind that the "take" of a graft will depend on

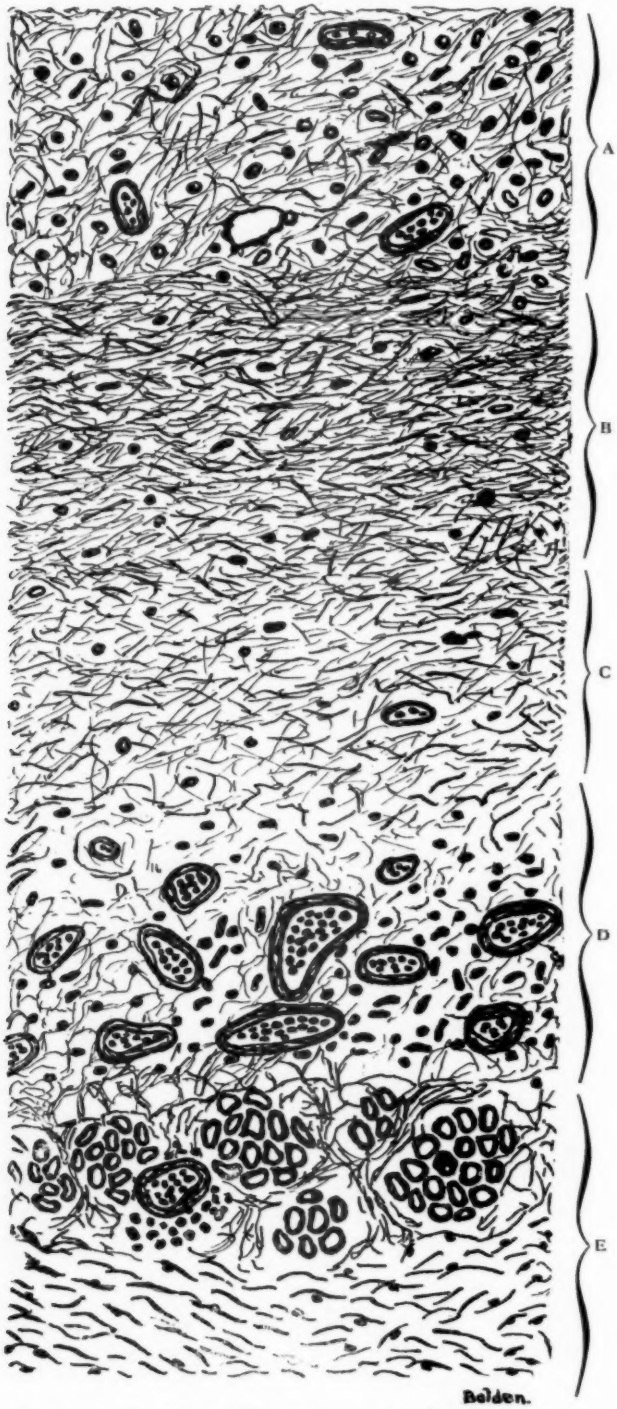


FIG. 2. Drawing illustrative of photomicrograph depicting successive layers in wound "fill"; A, granulation tissue; B, dense scar; C, less dense scar; D, rich vascular bed; E, subcutaneous tissue.

the balance between vascularization and infection, the former constructive and positive, the latter destructive and negative. Epithelial cells show remarkable vitality. "Beekman and Burdick at Bellevue Hospital, Staige Davis at Johns Hopkins, and Meleney at the Presbyterian Hospital have shown that pinch grafts may be applied to granulating surfaces even if there is a moderate amount of infection."[4] The desirable thing to insure success of a transplant is the provision of a bed that is the richest obtainable in vascularity with a minimum of infection. If infection is excessive the graft will die no matter how vascular the bed may be.

TECHNICAL CONSIDERATIONS

A wound site when properly prepared for the reception of an epithelial transplant presents, as previously stated, a surface that is dry, fibrin coated and of even topography. When the graft is applied to this bed it acquires an attachment, physicochemical in nature, which is almost immediate and due chiefly to the presence of fibrin. This initial fixity is particularly striking with the strip variety of graft, such as the Ollier-Thiersch, because of the broad surface area of attachment. The pinch graft, however, adheres, if not as obviously, just as adequately. For practical purposes, this initial fixity must be maintained, and in such a manner that apposition is firm, complete and without the interposition of foreign substance, as blood clot or ligature material. With good immobilization, vascularization will proceed uninterruptedly. If, for any reason whatever, immobilization should prove insecure and shifting of the graft occur, the capillary buds would be traumatized with the ensuing hemorrhage displacing the graft still further and ultimately interposing blood clot between the bed and the graft. Thus, vascularization would be seriously impaired and even impeded; and death of the transplant would ensue.

A vital factor in the immobilization of grafts is the amount of pressure to be applied. Too much pressure occludes the bed capillaries partially or completely, destroying their usefulness and making vascularization poor or impossible. Too little pressure gives ineffective immobilization. To strike the happy medium is essentially a matter of experience gained from repeated efforts. It is easy to see the significance of an even topography; mechanically it makes for even pressure in immobilization, yet, it is of even greater significance in the growth of the new epithelium on its bed. It is an accepted fact that epithelium, in its outgrowth, tends to follow the available base. Epithelial progress is much more rapid, and by far easier on a flat surface than on a nodular one. To quote Hartwell,[1] "One of the chief factors determining the rate of advance of an epithelial membrane is the type of base over which the membrane must progress."

RECEPTOR SITE

In the cleaning of a wound, after removal of crusts and debris, the use of a suitable antiseptic is indicated. Neutral acriflavin 1:5000, as recommended by Bennett, Blacklock and Browning,[5] has been in our experience more generally effective than any other in the treatment of extensive superficial wounds. Not only does it reduce surface infection rapidly, but it aids materially in promoting epithelization at the wound edge by forming an organic pellicle which adheres closely to the newly formed epithelium and mechanically helps the epithelial extension membrane to push its way forward over the wound. Three days before the operative procedure is attempted, acriflavin dressings are discontinued. The wound is cleaned again, the yellow acriflavin crusts removed from the edge, and the wound bathed with physiological salt solution. Injury to the capillary buds will produce bleeding and should be sedulously avoided. The peripheral skin is then cleansed with benzine, followed by alcohol, and finally ether is poured copiously over the wound and the surrounding skin. Then the wound receives a wet

dressing of equal parts of a saturated solution of boric acid and alcohol for forty-eight hours and the dressings are changed twice

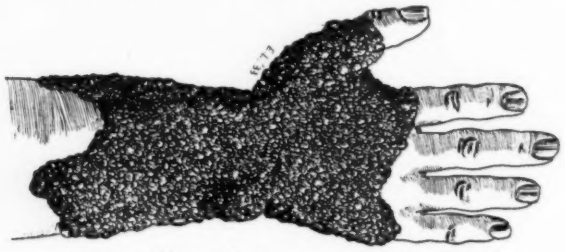


Fig. 3. Wound before preparation of bed.

daily during that time. After removal, a dressing of physiological salt solution is applied to the wound until the time of operation. At the operating table the wound should reveal a clean surface with pink compact granulations and no exudate (Fig. 3). The skin is painted with alcohol, followed by ether, drapes are applied, and the surgical excision begins. The first incision encircles the wound or portion of wound to be grafted and is carried down to the capillary bed. Reaching this vascular

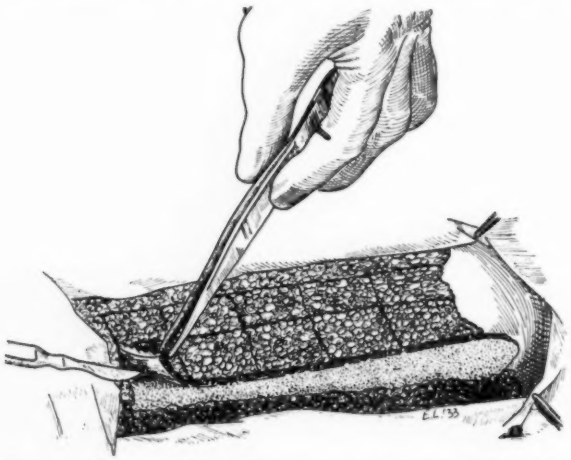


Fig. 4. Preparation of receptor site; wound gridironed, strips of granulation tissue excised down to rich capillary bed.

plane with ease and certainty, offers little difficulty; but when there is doubt, one should go beyond into normal tissue, whether it be fat or fascia. The capillary bed is found always immediately above normal tissue. The next step is the making of a sufficient number of parallel incisions, also down to the same plane. This gridironing of the wound facilitates excision. Under sharp dissection, each strip of granulation tissue is removed (Figs. 4 and 5). Bleeding

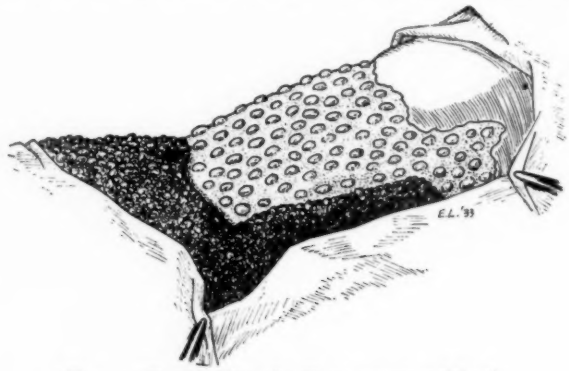


Fig. 5. Grafts "in situ" on prepared bed.

is controlled by pads, wrung dry from hot physiological salt solution, applied with firm and even pressure. No wiping should be done as this removes clots and initiates bleeding. Occasionally when one has gone beyond the capillary zone, a bleeding vessel is encountered, which, if it does not yield to crushing or torsion, or both, requires ligature. The very finest ligature material should be used, tied once and cut close. Every effort should be made to keep ligature material out of the bed. After bleeding has been controlled, the wound should be covered with pads moistened with physiological salt solution and attention then given to the donor site.

DONOR SITE

The donor site is chosen with consideration of the location of the wound, the character of skin desired, and the type of graft to be used. Certainly, in the use of a Blair split-skin graft[6] on the face, skin from the medial aspect of the arm is far more desirable than skin from the outer aspect of the thigh. However, for ordinary reconstructive work, where function is more important than appearance, the skin of the thigh is eminently satisfactory, especially from the anterior and outer surfaces. The donor site, shaved, and washed with green soap and water, and free from infection, particularly of follicular pustules, is painted with 3.5 per cent iodine in alcohol. This is

not allowed to dry but is immediately and completely removed with alcohol. The grafts are cut as ordinarily advised, for

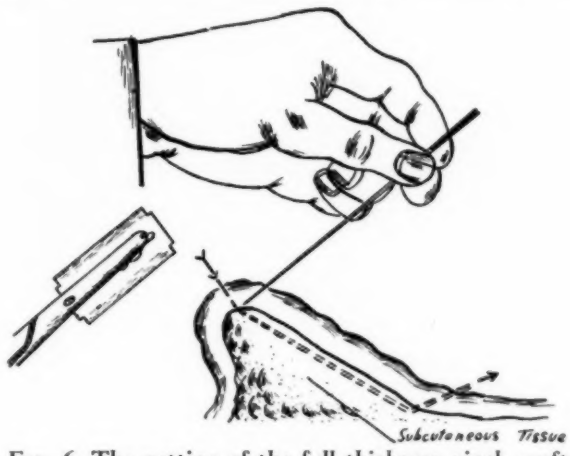


Fig. 6. The cutting of the full thickness pinch graft as described in text.

Reverdin pinch or Thiersch strip grafts. We have not found it necessary to use any distinctive apparatus in the cutting of grafts. We have modified the cutting of the Davis deep skin graft[7] in such a way that the entire pinch transplant is practically of full thickness. This is shown in the diagram (Fig. 6). With the needle in the left hand raising up a tuft of skin, the needle point and apex of the tuft towards the operator, an unequally inclined cone is formed so that the proximal segment is short and the distal segment long. This permits a cutting of the distal segment in such a manner that the portion of skin removed is almost whole thickness throughout (Fig. 7).

This type of graft gives an excellent integument, depending upon how closely together the grafts are placed. The full thickness pinch graft has found particular usefulness around joints where good firm resistant skin is desirable. I have used it in 3 cases in grafting skin over the patella; the end results have been excellent and there has been little cicatrization with this type of graft. It has given an integument that glides over the underlying structures with the freedom and pliancy of normal skin. These grafts may be applied immediately as each is cut, or they may be placed in physiological salt solution and then applied after all have been cut. The size of grafts must be taken into consideration.

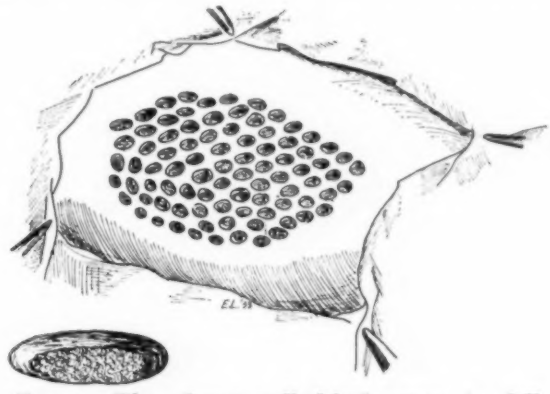


Fig. 7. The almost cylindrical crater in full thickness pinch graft.

The pinch type of graft that I employ is of good size, with a diameter usually 5 to 9 mm. This is much larger than the classical Reverdin graft[8] or the Davis deep skin graft.[7]

I have found little use for the true Reverdin[8] and Thiersch grafts[9] with their very small portion of corium or derma, because scarring is more frequent following the use of this thin type of graft.

In the Thiersch grafts nothing but the epidermis or the epithelial elements are used. The contraction after an injury to the skin of the face, for instance, is not in the epithelial elements of the skin but in the connective tissue that underlies the epithelium. In other words, the contraction lies in what corresponds to the corium which is composed largely of connective tissue and on which rests the epithelial layer. If, in the healing process, this is made up of scar tissue, particularly of the dense scar tissue that follows a burn, contraction deformity will probably result even though the surface may be covered by a healthy epithelium. It is contraction in this cut epithelial layer that produces the striking deformities following burns of the face or hands with the eversion and twisting of features . . . In order to avoid this it is necessary to use the whole skin or nearly the whole skin, which contains not only the epidermis but normal healthy corium that does not contract.[11]

The Thiersch graft is excellent for plane surfaces where closure is the sole aim but

not where scarring might produce deformity or interfere with function and therefore be of serious concern. A thick graft, like the Blair split skin graft[6] with a greater portion of corium, is far more satisfactory than the Thiersch,[9] just as the Davis deep skin[7] and the full thickness pinch grafts are superior to the Reverdin.[8] The ideal is the Wolfe graft but its range of applicability is limited and its success too precarious for general use.

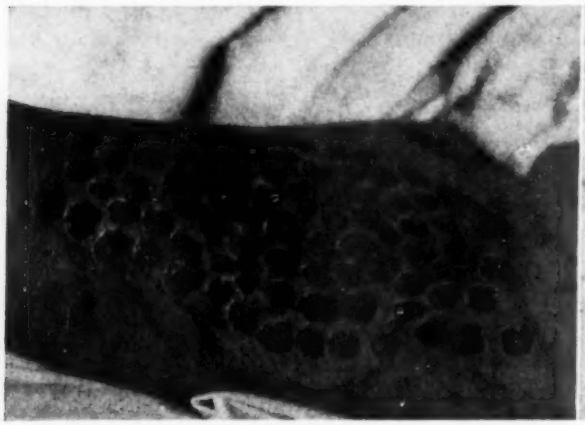


FIG. 8. Tannic acid eschars formed at wounds of donor site.

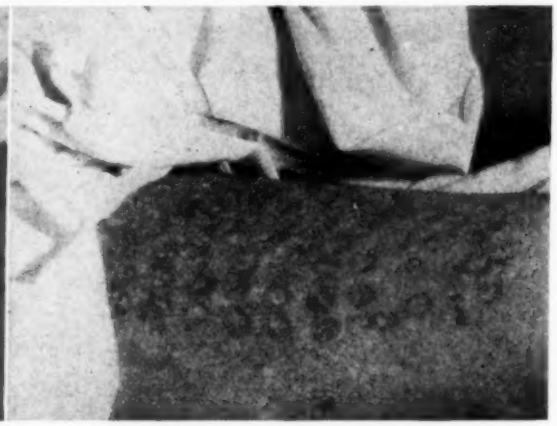


FIG. 9. Healed areas after falling away of eschars.

The dressing of the grafts requires great attention to details. Immobilization should be secure in order to prevent shifting of the transplants. When the grafts have all been appropriately placed, a fenestrated paraffin gauze called "parawax" is firmly and evenly applied. Over this is placed a number of layers of gauze, wrung dry from warm physiological salt solution, and of equal thickness throughout. Then a moderately thick fluffy pad of non-absorbent cotton is superimposed, followed by a final layer of gauze. The entire dressing is then snugly bandaged with a view to adequate pressure; and if feasible, the part is splinted in plaster. When the patient is returned to bed the grafted part is placed under a cradle and the temperature maintained at 80 to 85 degrees Fahrenheit.

In the case of strip grafts such as the Thiersch,[9] dressings are untouched for seven days. In the case of any type of pinch grafts, dressings are undisturbed for eleven days. All dressings are removed down to the paraffin gauze. There may be a considerable amount of purulent exudate, somewhat malodorous, saturating the gauze dressings but this should not cause concern. If the exudate is washed away from the "parawax" with normal physiological salt solution, the grafts are seen closely, and sometimes, deeply embedded, and surrounded by bright red granulation tissue. The use of peroxide makes the removal of the "parawax" easy and without disturbing the underlying transplants. The partially healed wound is revealed as a mosaic of epithelial plaques and interlacing pink granulations. To produce rapid wound closure, we have used a neutral acriflavin 1:5000 as a wet dressing, and have sought to maintain the granulation tissue about the grafts, flat, compact, and below the advancing epithelial edge so that the amoeboid extension of cells from the periphery of the graft meets no mechanical impediment to its outgrowth. Neutral acriflavin 1:5000 has been preferred because it is innocuous to the growing epithelium, antiseptic, and, by forming a closely applied organic pellicle, aids materially in the mechanical progress of the newly formed epithelial cells.

TREATMENT OF WOUNDS OF DONOR SITE

In the treatment of wounds of the donor site, I instituted a method in 1930, of which,

to my knowledge, no mention was made in surgical literature up to that time. Since then, in over 200 cases embracing a wide variety of grafts, thick, medium, and thin, the classical Reverdin,[8] Davis deep skin and full thickness pinch,[7] the Ollier-Thiersch,[9] and the Blair split skin graft,[6] I have treated wounds of the donor site with tannic acid (Figs. 8 and 9). The results have been uniformly good and especially gratifying in the pinch type of graft. Tannic acid has been used in the form of a water soluble jelly containing five per cent tannic acid and merthiolate 1:5000. The merthiolate has been desirable because of its germicidal qualities. Regarding this, Fowler and Martin,[11] in their bactericidal studies, found that while tannic acid in the usual 5 per cent strength is not sufficiently germicidal and may in itself be the agent of conveyance of bacteria, yet, with the addition of merthiolate 1:10,000, it is lethal to all organisms within twenty-four hours. Stronger solutions of tannic acid, such as 10 per cent or 20 per cent, are definitely bactericidal; but, as has been established by Seeger,[12] these are too highly astringent and tend to cause swelling and edema of the tissues. For this reason, solutions of this strength are held undesirable and accordingly are not recommended for use.

From the patient's standpoint, if applied to the wounds of the donor site when he is conscious of sensation, analgesia is evident within one-half hour of its application. When the eschars are fully formed (Fig. 8), pain is completely absent and the patient is comfortable and entirely at ease, since no further bothersome dressings will be necessary. From the point of view of the surgeon, the application is simple, sure in effect and practically ensures absence of infection. In my entire series there was not a single instance of infection. Scarring has been greatly minimized and in most cases when the eschar fell away the wound was completely healed (Fig. 9).

Procedure. The wounds of the donor site must be completely dry before the application of tannic acid jelly. There must be neither free bleeding nor continued ooze. Moreover, it is desirable that gross clots

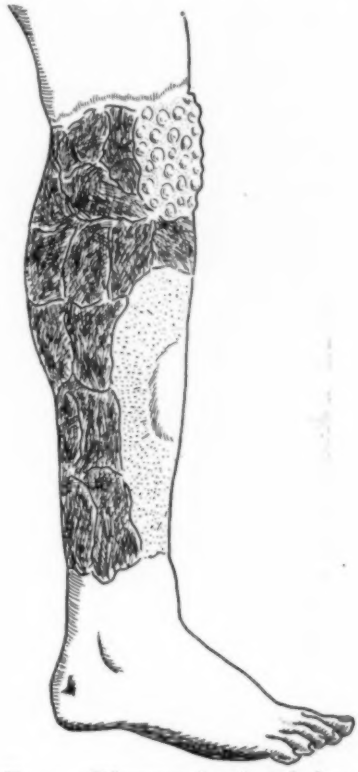


Fig. 10. Diagram showing choice of grafts in Case 1.

should be removed from the wound and skin margins. A liberal application of the jelly is made to the wounds, which may be bandaged or left exposed.

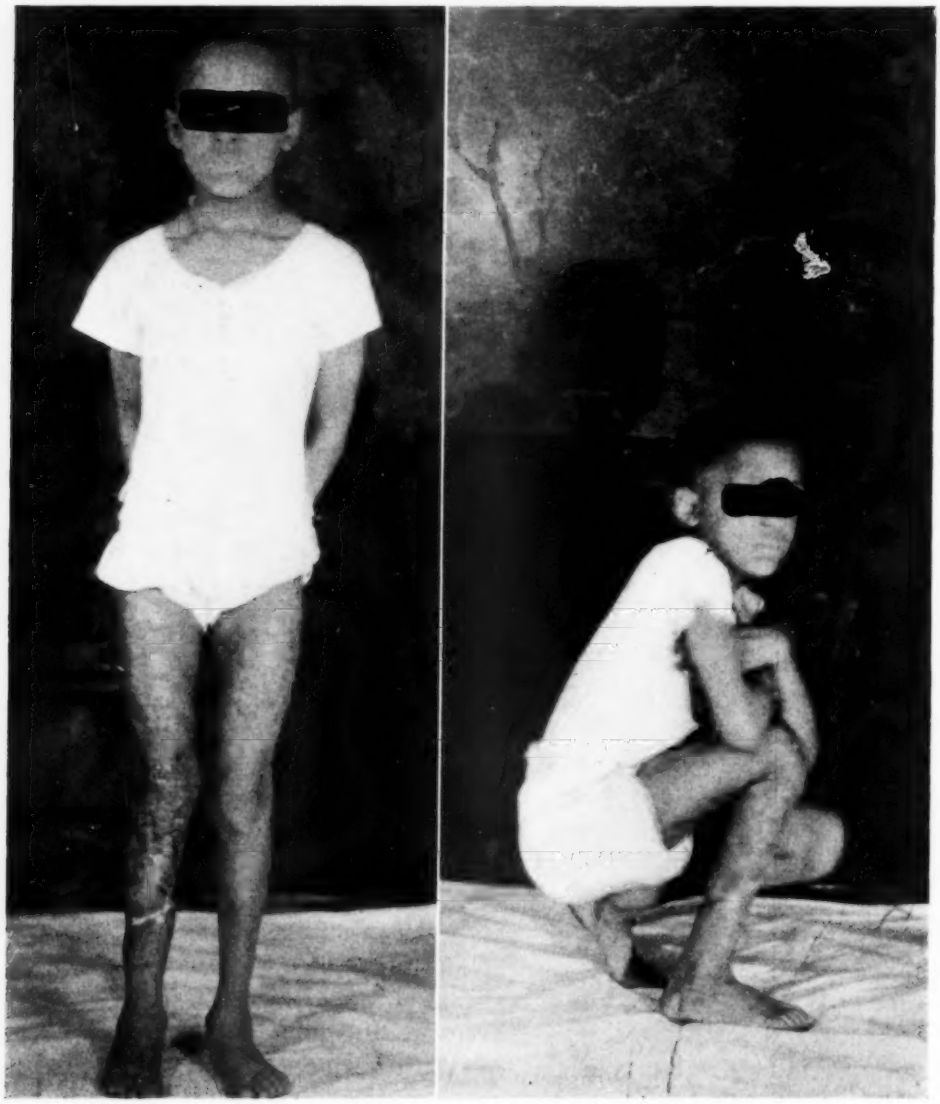
I prefer exposure because the tanned membrane forms under direct observation and more quickly; it remains undisturbed by dressings, which stiffen and are often so adherent to the partially or completely formed eschar that removal without injury to the eschar is not only difficult but sometimes impossible.

Six hours after the first application, another is made. This is usually sufficient; and, within six hours from this time, the desired firm, rigid, and adherent eschar will have formed.

Care should be taken by both patient and attendant to avoid any trauma that might impair the integrity of the eschar or dislodge any portion of it from its bed.

Injury of this sort would predispose to infection, since this is akin to wounding the skin. Eventually, the eschars fall away spontaneously leaving a healed area with a minimum of scarring.

It is significant that, in the case of a large number of colored patients, keloid formation has developed at a later stage following the healing, even as long as six months after.



A B

FIG. 11. Case 1. Full thickness pinch grafts used over patella. Note functional excellence.

ANESTHESIA

We have found general anesthesia to be especially desirable for this work. However, when there have been definite surgical indications, local or regional anesthesia has been employed. Since the operative procedure is often quite prolonged because of due attention to details, and since blood loss at the receptor site may be sometimes inordinate, it is probably wise to subject the patient to as little anesthetic strain as possible by avoiding too deep narcosis. Such has been our habit.

CASE REPORTS

CASE 1. S. W., colored boy, aged eight years, was admitted to the hospital June 8,

1932, in deep shock, pulseless, unobtainable blood pressure and a history of having been hit by a truck. Treatment for shock was immediately instituted. When the patient reacted examination revealed the following injuries: (1) a 3½ inch laceration of the scalp down to bone in the right frontoparietal region; (2) an avulsion of the skin from the lower thigh, knee and leg with laceration of the bellies of the gastrocnemius and soleus muscles. The skin avulsion extended from just above the upper border of the patella to 1½ inches above the external malleolus, completely encircling the leg except for an attachment of skin over the crest and anterior surface of the tibia; and (3) a compound comminuted fracture of the tibia and fibula in the middle third with good position of fragments.

A B

Fig. 12. Case 11. The entire anterior, lateral and medial aspects of left from just above patella to just above malleoli grafted. Full thickness pinch grafts were applied to patella; thick plaques of skin, including most of derma, applied to rest of wound. A, anterior aspect; B, lateral aspect.

Most of the avulsed skin necrosed and left an extensive wound surface for repair so that on August 18 the patient presented a wound

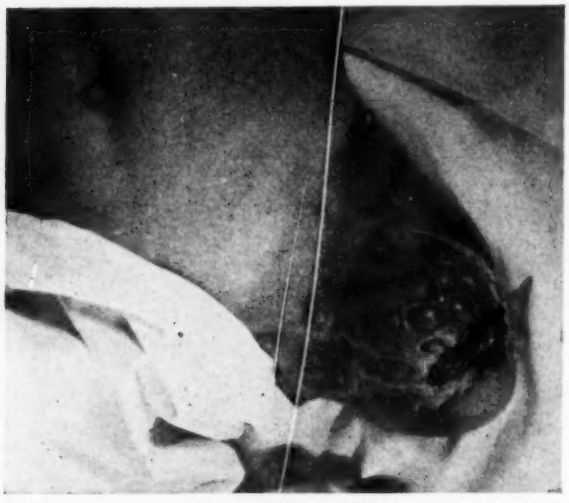


FIG. 13. Case III. Full thickness pinch grafts for large epithelial defect in stump of a high amputation for gas grangrene complicating extensive avulsion and crushing injury of the soft tissues.

extending from just above the level of the upper margin of the patella to about 2 inches above the external malleolus completely around the leg except for the anterior plaque of skin over the tibia. The wound was clean and considered suitable for grafting at this time. Full thickness pinch grafts, about 7 mm. in diameter, were applied 3 to 4 mm. apart, to the wound over the patella and just beyond its margins. On the eleventh day following this procedure the dressings were cut down and the site of operation inspected. The dressings closest to the wound were foul smelling and saturated with thick and partially caked purulent discharge. The "parawax" was left "in situ" and the purulent matter washed away with physiological salt solution. The grafts were then seen as healthy looking "takes" with a thin whitish epidermal membrane extending from the margin of each graft. Neutral acriflavin 1:5000 moist dressings were applied to the wound to prepare it for the second grafting.

In three weeks the second skin graft was made over the upper portion of the leg on the posterior, medial and lateral aspects. The skin used in this graft was applied in the form of rectangular strips containing the greatest portion, but not all, of corium. The wound was inspected on the seventh day postoperative and all of the grafts were found to be alive.

Seventeen days later the third skin graft was made over the remaining area. Plaques of skin were employed just as in previous operations. All were "takes."

October 28, the leg was completely covered with epithelium and the job was considered finished (Fig. 10).

Ever since the first operation, motion had been instituted at the knee. At first passively, and then very gradually and with encouragement, active motion was obtained so that by the time there was complete epithelization of the wound, the child had practically full function at the knee.

About two weeks after the patient's discharge from the hospital, the grafted skin was examined. The best integument was that over the patella which could be picked up by the fingers and made to glide with freedom over the patella. The skin was apparently free from cicatrization and quite firm and resistant (Fig. 11, A and B). Sensitivity to pain and temperature, however, was slight, if at all. The strip grafts gave excellent skin but not as pliant an integument as the full thickness pinches.

This child has been seen recently; he has perfect function and the cosmetic result is much better than it was a year previously.

CASE II. W. W., a colored woman, aged twenty-nine years, was admitted to hospital July 8, 1935, in shock with a barely preceptible pulse, having been struck by an automobile. The findings were as follows: "The left leg presents an avulsion of skin extending from just above patella to dorsum of foot and the external malleolus, the tibialis anticus muscle lacerated and half of its belly avulsed. The skin has been torn away on both sides of the tibial crests and hangs loosely in folds. Patient is stuporous and aroused with difficulty."

Treatment instituted locally was debridement and partial suture of the skin, insertion of drainage tubes and wet potassium permanganate 1:10,000 dressings with continuous drip, and the foot kept elevated.

Within six weeks the leg was granulating nicely over an area extending from the upper level of the patella down to the level of the external malleolus on all aspects of the leg with the exception of the central portion of the posterior surface which retained normal skin. The wound was now considered clean and ready for grafting. The patient, however, had a red blood cell count of 2,200,000 and a 54 per cent

hemoglobin. A blood transfusion of 500 c.c. whole blood was given four hours before operation.

A B

Fig. 14. Result of flame burn, shows "takes" on shoulder and upper axilla. Preoperative immobilization of the extremity to the trunk was accomplished by means of a plaster body spica with full exposure of the area to be grafted; this facilitated the operative procedure.

At operation, the granulation tissue was excised from the patella area in preparation of a bed for the grafts. The patient lost a considerable amount of blood despite our best efforts at hemostasis and went into shock. The operation was stopped, the patient treated with saline infusion, warm blankets, and placed into the Trendelenburg position. When the patient reacted, she was returned to the ward.

The following day the patient was given a small transfusion of 200 c.c. of citrated blood, taken to the operating room, and full thickness pinch grafts were applied to the bed that had been prepared the day before. On the eleventh postoperative day the grafts were inspected; all were "takes."

The next day the medial aspect of the wound was grafted with split skin transplants. The patient's anemia still profound, red cells 2,200,000 with hemoglobin 45 per cent. She was given calcium preoperatively, 200 c.c. of citrated blood the day before operation, and during the operation kept on continuous intravenous saline drip. The seventh day postoperatively the wound was inspected; all were "takes."

November 18, 1935, a portion of the anterior aspect of the wound of the upper left leg was grafted with split skin transplants with the same precautions taken to fortify patient, with small transfusion of 200 c.c. of citrated blood before operation and intravenous saline drip at time of operation. She reacted well. Seven days postoperatively the wound was inspected and all were "takes."

Six weeks later the exuberant granulation tissue was removed from the rest of wound area and between the transplants and a week after this the epithelial strips applied to the anterior aspect of the wound of the lower leg. The wound was inspected on the seventh day and all were "takes."

February 7, 1936 a small portion of skin was applied to a small wound area on the anterior aspect of the leg to hasten wound closure.

During the patient's stay in the hospital she developed a flexion contracture at the knee that might have been avoided in some degree

Fig. 15. In May, 1930, this patient had a severe burn with extensive skin loss. The leg was grafted in the fifth and seventh weeks following the burn with successful result and no resulting contracture, so that the patient walks well. At the present time (1937) the grafted skin is of excellent texture with tessellations obvious only on close inspection. There is almost complete return of sensation to the grafted area; the pain sense is only slightly less acute than in the normal surrounding skin; the temperature sense is apparently normal.

with a bit more cooperation on her part. However, she was discharged on April 5, 1936, nine months after the accident with a fair degree of flexion mobility at knee but with inability to get full extension (Fig. 12, A and B).

Case III. R. B., aged six years, was knocked down and dragged a distance of fifty feet by a truck and was in shock when admitted to the hospital March 14, 1936. His injuries comprised an avulsion of the skin of the entire left lower extremity from just below the anterior superior spine to the dorsum of the foot. The only area of attachment of skin was to the posteromedial aspect of the upper one-fourth of the thigh. There was also an intra-articular fracture of the lower end of the femur. The muscles were badly lacerated and contused. The fascial planes were opened, and extensive debridement with removal of the skin that was obviously devitalized, and dakinization were done. Two days later because of gas gangrene, disarticulation at knee was done. This did not suffice and necessitated a high amputation of the thigh at the level of the apex of Scarpa's triangle.

Two months later the amputation stump was devoid of skin on the anterior, medial and lateral aspects extending to the groin. The wound revealed healthy pink compact granulations and was considered ready for grafting.

A week later full thickness pinch grafts, 8 mm. in diameter were applied to the area about 3 to 4 mm. apart and when the dressings were removed in ten days all grafts were "takes" and rather deeply embedded in granulations, which were cut down by a silver nitrate stick.

June 12, 1936, the wound area was completely closed except for an area over the end of the bone (Fig. 13).

COMMENT

Successful skin grafting may be regarded as the integration of a number of steps, each competently and sequentially followed. At first there is the cleaning of the wound site; then the preparation of the bed; and finally the cutting, application and immobilization of grafts. Each step is an indispensable link and a break in any part of the chain may cause disaster.

The patient with an extensive loss of skin presents a distinct problem. Time induces constitutional debility because of infection, toxic absorption and fluid loss. To avoid this morbid state with its tragic consequences of exhaustion and general sepsis, early skin grafting becomes a therapeutic urgency.

In this connection it is worthy of note that high fever is not a contra-indication to operation, for in the majority of our extensive burns which were grafted, the patients ran a temperature range of 100 to 103°F. at time of operation.

It is advisable that every effort is made to fortify the patient before and after operation, if necessary, by adequate blood transfusion. Judgment, too, should be exercised in the extensiveness of the operation in view of the danger of a sometimes inordinate blood loss incident to the exci-

sion of scar tissue in the preparation of the graft bed.

Fixation of grafts by dressings is done with difficulty at certain sites, notably the neck and axilla. There is no assurance that when initially fixed these small and unsutured transplants will remain in place long enough for the capillary buds to grow into them. From our own experience it has seemed that since postoperative mobility is the disturbing factor in this instance, preoperative immobilization of the part with full exposure of the wound should be done when so indicated. In our work, plaster used with direction, purpose and ingenuity has sufficed for immobilization. For example, in grafting the anterior axillary fold and medial wall of the axilla, we have employed a body spica of plaster holding the arm in 120 degrees abduction and with the operative site fully exposed. In grafting the anterior and lateral portion of the neck following a burn, to avoid mentothoracic adhesions, the head was fixed to the body by an encircling paster cap holding the head back in extension and immovably connected to a plaster body spica by an iron stave. This simplified the task immensely.

CONCLUSION

1. In the preparation of a bed for the reception of grafts, the complete excision of granulation tissue down to the capillary stratum is indicated, because

(a) A bed of maximum vascularity is obtained; and (b) A scar is eliminated, thereby lessening the danger of contraction deformity.

2. Whenever possible thick grafts containing most or all of corium should be used.

(a) The full thickness pinch graft from the point of view of function produces the best type of integument. It produces not only the most serviceable skin, but all factors considered, closes an area more quickly than any other type of transplant.

3. Tannic acid is highly efficacious in the treatment of wounds of the donor site.

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CASE REPORTS

HEMORRHAGE INTO HYDATID OF MORGAGNI SIMULATING ACUTE APPENDICITIS

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TO 6 cases of hemorrhage into or torsion of the hydatid of Morgagni so far reported in the literature, we wish to add 2 cases of our own.

CASE 1. A girl, aged thirteen years, was first seen by her family physician at 9:00 A.M., July 2, 1936. The patient told her physician that she was awakened at 1:00 A.M. by a generalized abdominal discomfort which gradually localized in the right lower quadrant. This was accompanied by some nausea and vomiting. An enema was given which, while effective, did not relieve the pain. Physical findings at this time revealed definite tenderness and rigidity over McBurney's point. The white blood count was 14,000 of which 78 per cent were polymorphonuclears.

At 4:00 P.M. when I first saw the patient essentially the same history was obtained. On physical examination, in addition to the tenderness and rigidity mentioned, there was marked rebound tenderness over the right side of the abdomen. At this time the leucocyte count was 14,600 of which 80 per cent were polymorphonuclears.

A diagnosis of acute appendicitis was made and immediate operation was advised.

Operation. The abdomen was opened through a right rectus incision in the area between the symphysis pubis and umbilicus. A small amount of serosanguinous fluid was present in the peritoneal cavity. Though the appendix showed no marked pathology it was removed. Since the clinical picture could not be explained by the slight pathology in the appendix, the pelvic organs were examined. The right ovary was found to be cystic and enlarged to about 4.5 by 3.5 cm. Aspiration showed that it contained a straw colored fluid. Attached to the right salpinx was a small mass which had the appearance of an organized blood clot. It was removed. The uterus, left tube and ovary were normal on inspection.

Pathology. "The appendix measured 6.2 cm. by 0.7 cm. Grossly the peritoneal surface is smooth, slightly injected, with the vessels somewhat dilated. Section shows the lumen to be filled with soft fecal material. The mucosa is pale red in color and free from any marked exudate.

"The second specimen is a dumb-bell shaped structure 3 cm. in length, which has the appearance of a blood clot. Its isthmus has a diameter of 4 mm. One end has a diameter of 1.2 by 0.7 cm. The other end is 1.2 by 0.7 cm. To the larger end a membrane-like structure is attached which simulates fetal membrane, with some softer red tissue. There is a small cyst containing watery fluid at the smaller end.

"Microscopic section of the hemorrhagic structure reveals patched erythrocytes among which a few strands of smooth muscle, connective tissue, and in places supporting blood vessels are noted. Parovarium structures are also present in this. These are characterized here by a narrow strip of smooth muscle covered over on the inner surface by a single layer of columnar epithelial cells. One of these structures is dilated and forms a cyst in which there is flattening of the lining epithelium. The absence of blood pigment signifies that the haemorrhage is of very recent origin.

"Microscopic examination of sections from three different portions of the appendix, reveals slight fibrous thickening of the serosa, in which there is also manifest a slight edema and a distention of the venules with blood. The muscularis is thin in places, but the submucosa and the mucosa are unaltered.

"The pathological diagnosis is hemorrhage into the tissue of the broad ligament about the hydatid of Morgagni, probably due to torsion."

Case II. R. J. H., female, aged thirty-four years, was seen October 8, 1936, complaining of palpitation and tachycardia. At times, the patient stated she had severe aching pain in the left lower abdomen, aggravated by intercourse.

Physical examination at the time of her first visit was negative except for a small, tender mass in the left adnexal region and tenderness on palpation over the left lower quadrant.

On October 30 the patient complained of severe left sided, cramp-like pain and backache. Her menses were due that day. Pelvic examination revealed the cystic mass had increased in size and was quite tender. A tentative diagnosis of torsion of the pedicle of the left cystic ovary, or an endometriosis of left adnexa was made.

At operation on November 16 there was a retroverted uterus and a hydatid of Morgagni on the left, which was enlarged and cystic with a long pedicle which showed a double twist in counter clockwise direction.

The uterus was suspended and the hydatid of Morgagni was removed.

The patient has been entirely free from pain for six months.

It is interesting to note that 3 of the 6 instances found in the literature were diagnosed preoperatively as acute appendicitis.

Table 1 gives a summary of the cases of torsion which were diagnosed as acute appendicitis.

The other cases were diagnosed as either an abnormal pregnancy or pregnancy complicated by torsion ovarian cyst.

Tractenberg's patient, a married woman, twenty-nine years of age, was diagnosed as a ruptured left ectopic pregnancy. There was acute pain on the left side. The leucocyte count was 13,500 with 80 per cent polymorphonclears. The menses were irregular.

At operation the appendix was normal, both ovaries were cystic, and a hydatid of Morgagni with torsion and hemorrhage was found on the left.

Andrew's patient was thirty-six years of age, married, and at that time six months pregnant. The preoperative diagnosis was torsion of the pedicle of a cystic ovary. There was marked distention. At operation the abdomen contained much fluid. Torsion of a hydatid of Morgagni was found.

Abernathy's patient was a married woman, thirty-three years of age, with a history of incomplete abortion. There was abdominal pain two months later. A dilatation and curettage followed by an exploratory laparotomy revealed the products of conception, an old chronic appendicitis; a hydatid of Morgagni, right, thrombosed from torsion and bilateral cystic ovaries.

The comparatively rare condition of torsion of, or hemorrhage into the hydatid of Morgagni does cause acute abdominal

TABLE I

Author	Age	Site of Pain	Menstrual History	Preoperative Diagnosis	Appendix	Ovaries	Fluid in Abdomen	Torsion of Hydatid
Waters	18	Left lower quadrant	Irregular	Acute appendicitis, left sided	Normal	Both cystic	Yes	Left
McDermott	16	Right lower quadrant	Irregular	Acute appendicitis	Normal	Right cystic	Yes	Right
Heinrick	15	Right lower quadrant	Irregular	Acute appendicitis	Normal	Both cystic	Yes	Right
Zener	13	Right lower quadrant	Irregular	Acute appendicitis	Normal	Right cystic	Yes	Right

symptoms. Finally, where the preoperative diagnosis of lower abdominal conditions is not substantiated at operation, this condition should be kept in mind and search made for it, as it is easily overlooked.

SUMMARY

Two cases of torsion of and hemorrhage into the hydatid of Morgagni are presented. The clinical picture seen is that of acute appendicitis. A review of the literature reveals 3 similar cases and 3 cases where the condition complicated pregnancy or simulated abnormalities of pregnancy.

Emphasis on exploration of the pelvis in all cases is stressed.

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Acute empyema or suppurative pleurisy is the commonest surgical lesion of the chest, but in proportion to the frequency of its occurrence it is, perhaps, the lesion which is most often badly treated, or neglected.
From—"Preoperative and Postoperative Treatment" by Robert L. Mason, M.D.

In the diagnosis of abdominal tumors Bishop Butler's maxim that "probability is the rule of life" is particularly true, and the cocksureness of the clinical physician, who formerly had to dread only the mortifying disclosures of the post-mortem room, is now wisely tempered when the surgeon can so promptly and safely decide upon the nature of an obscure case.
From—"Lectures on the Diagnosis of Abdominal Tumors." Reprinted from The New York Medical Journal, 1894.

UNCONTROLLABLE HEMORRHAGE OF PROSTATIC ENLARGEMENT

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HEMATURIA is of frequent occurrence in prostatism, being present in 16 per cent of the cases.[1] Young[2] explains the infrequency of hematuria in prostatic carcinoma by, "the fact that carcinoma of the prostate does not invade the bladder except in a small proportion of cases, but is retrovesical and pelvic rather than intravesical." It is not unusual therefore to find that all reported cases of uncontrollable hemorrhage in prostatic enlargement are limited to benign hypertrophy.

Shivers[3] in 1935 collected 26 cases of uncontrollable hemorrhage from benign prostatic enlargement from the literature and added one of his own.

So far the etiological factors mentioned are of local origin namely:

1. Dilated vessels on the surface of the prostate gland;
2. Ulcerations on the gland surface;
3. Generalized prostatic congestion with capillary bleeding; and
4. Combinations of the above.

The bleeding is usually from the middle lobe. According to Shivers,[3] the veins found over the intravesical surface of the middle lobe are submucous in type and either emerge from the sides of the prostate or about the posterior portion of the vesical outlet. These veins spread over the surface of the lobe as it hypertrophies and pushes its way through the internal sphincter. Contracture of the internal sphincter about the neck of the middle lobe together with pressure of this lobe against the mucosa, causes a slowing of the circulation and results in varicose vessels.

Ulcerations may be caused by infection either before or after instrumentation. Lett[4] cited a case in which a calculus caused an ulcer on the posterior surface of the prostate ¾ inch in diameter. The patient "after losing a considerable amount of blood on successive days, arrived in clot retention."

To the purely local causes listed we wish to add one of a more general nature, namely hypertension associated with benign prostatic hypertrophy.

The patient, aged sixty-seven years, was a large, well proportioned, plethoric Hebrew weighing 220 pounds. He was referred to us on April 10, 1936, because of hematuria. His first attack of hematuria, total in character, was in January, 1936. At that time it continued for two days and then ceased. When seen on April 10 the patient had a total hematuria and passed numerous small clots with each voiding. He also complained of some "stinging" at the end of the penis before urination. Nocturia, causing the patient to void one to two times, had been present for five years. Lately there has been some slowness in starting the stream. The patient stated that he had a high blood pressure for which he was undergoing treatment.

Physical examination of the upper abdomen revealed nothing unusual. A left inguinal herniotomy scar and a right indirect inguinal hernia were present.

On rectal digital examination the prostate was found to be only moderately enlarged, and elastic in consistency. The median sulcus was shallow and the lateral sulci somewhat deepened.

After the patient had voided a 14 F. soft rubber catheter was passed to the bladder, and 28 c.c. of bloody urine obtained. Lengthening of the urethra could not be definitely determined. The bladder was then irrigated with warm boric acid.

Vesical neoplasm had also to be considered, for, though symptoms suggestive of prostatism were present, the prostate per rectum revealed no remarkable change. Moreover definite evidence of urethral lengthening could not be established, and the residual urine was slight. Therefore it was decided to cystoscope the

patient in the office the next day as the patient refused hospitalization.

On April 11 the hematuria seemed to have increased. At 3.30 P.M. a No. 21 Brown-Buerger convex cystoscope was easily passed to the bladder. No obstruction was encountered, but in order to allow the irrigating fluid to flow into the bladder the convexity of the scope had to be turned upward to 12 o'clock. Only a very poor view of the interior of the bladder was obtained, due to bleeding, which appeared to be coming from the region of the bladder neck. One could determine however that there was marked intravesical intrusion of the middle and both lateral lobes. The ureteral orifices could not be seen, nor could the remaining portion of the bladder be visualized.

The cystoscope was withdrawn gently. A few moments later drops of blood made their appearance at the external meatus.

A soft rubber two-holed 18 F. catheter was passed to the bladder to determine the seriousness of the hemorrhage. The bleeding continued. Drops of blood dripped fairly rapidly from the end of the catheter. Finally, one gained the impression that blood clots were accumulating in the bladder. The catheter was withdrawn.

The seriousness of the condition was impressed on the patient, and immediate operation advised. No little difficulty and loss of time was experienced in persuading the patient to be hospitalized. Because of religious duties he wished the operation postponed two days.

At the time of operation, 6:55 P.M., the bladder was distended to two fingerbreadths above the umbilicus. Under general anesthesia the usual suprapubic cystotomy incision was made and carried down to the bladder. A small incision was made through the bladder wall to admit the suction tip. The suction apparatus soon became useless due to clots. The incision was enlarged and the clots removed manually.

The bleeding was seen to be coming from the region of the prostate gland. It was a generalized oozing rather than from any individual vessel. The prostatic urethra and intravesical prostatic collar were packed with 1½ inch plain gauze. The bleeding showed no sign of abatement. A partially distended Pilcher bag was then tried. As the danger of tearing through the gland was too great, the bag was removed by snipping the tubing intravesically. The question of enucleation of the prostate was next considered. It was discarded for the patient was in no condition to withstand so radical a procedure and only a larger bleeding area would result. This area would be too large to be covered by a large size Pilcher bag, and packing in this instance would be of less value with the prostate removed. Therefore the prostatic urethra and intravesical prostatic collar were repacked with plain gauze. The bladder opening was closed around a large rubber tube. Suction was necessary to keep the field dry. The patient was returned to the ward in fair condition.

His pulse rate at the beginning of the operation was irregular, of good volume, and 102 per minute. At the close of operation the pulse rate was more irregular, weak, and 140 per minute. Shortly after cessation of anesthesia the patient became very violent and had to be restrained, being finally controlled by morphine. Bleeding continued. The dressings were changed frequently and arrangements were made for transfusion. A few hours later, it was noted that the bleeding was much less. Just before midnight the bleeding had practically ceased. The blood pressure at this time was 170/120 and the pulse rate 80 per minute.

Blood examination the next day showed 4,400,000 red blood cells, 20,700 white cells and a hemoglobin of 84 per cent (Osgood Haskins).

On April 29 the prostate was enucleated through the suprapubic opening. Only the usual amount of bleeding was encountered. The prostate shelled easily. The blood pressure at this time was 150/80 and the pulse rate 80 per minute, though still irregular, but the irregularity was not so marked as the patient had been placed on digitalis therapy.

The patient made an uneventful recovery and was discharged dry suprapubically on May 14, 1936.

The removed prostatic tissue weighed 79 grams. Pathological examination revealed benign glandular hyperplasia.

On May 24 the patient again hemorrhaged per urethra. He was sent to the hospital immediately. The blood pressure at this time was 260/140. Prior to operation the bladder was two fingerbreadths below the umbilicus.

The bladder was opened in the region of the old incision and after evacuation of bloody urine and clots, the prostatic bed was packed with gauze.

The patient made an uneventful recovery and was discharged on June 18, 1936. Since then the patient has had no further urinary trouble. He has no residual urine.

Examination of the patient's circulatory system revealed the presence of advanced arteriosclerosis. The heart was markedly enlarged, and a rather severe auricular fibrillation was present.

Later information from the patient's physician disclosed that a blood pressure of 270/160 and higher had been recorded. In a few instances systolic pressures were obtained, which were not registerable on the ordinary sphygmomanometer, which registers up to and including pressures of 300 systolic.

That the severity of the hemorrhage was mainly due to the marked hypertension is clearly illustrated by the fact that the bleeding which continued despite suprapubic drainage and packing, ceased when the blood pressure was reduced to 170/120. Enucleation of the prostate, when the blood pressure was 150/80, with only the usual amount of bleeding, lends further emphasis. When the patient again became active, nearly a month after enucleation, hemorrhage from the prostatic bed resulted. Though some infection was present in the prostatic bed the blood pressure registered 260/140 and was we believe the chief contributing factor. The absence of further bleeding in this area is easily explained by the healing of the operative site and a maintainence of a systolic pressure below 200.

In 15 of the previously reported cases it was necessary to enucleate the prostate to save the patient's life.[3] In the instance reported enucleation, though discussed during the first operation, was discarded as the patient would not have survived the shock of so radical a procedure. One would have obtained only a larger bleeding area with a correspondingly larger uncontrollable hemorrhage. The later course of events sufficiently emphasizes these facts.

CONCLUSIONS

1. This case emphasizes the importance of preliminary hospitalization with bed rest for control of the blood pressure in prostatic hemorrhage associated with hypertension. In this instance the patient refused to cooperate.

2. Once severe hemorrhage has set in only cystotomy and packing of the prostatic area should be attempted. An extra large rubber tube should be left in the bladder for evacuation of blood clots.

3. Enucleation of the prostate as an emergency measure is contra-indicated.

4. Later, after the patient has recuperated from the effects of the initial operation, and the blood pressure is within reasonable limits, suprapubic enucleation of the prostate is the method of choice, and will be productive of better results than any other method.

5. When the patient is again ambulatory, his activities should be limited, and an effort should be made to keep the blood pressure in check. In our instance the patient cooperated very poorly and secondary hemorrhage resulted.

6. With complete healing of the prostatic bed after enucleation, the possibility of hemorrhage from this area is probably remote.

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CONGENITAL COXA VARA OCCURRING IN IDENTICAL TWINS*

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THE occurrence of congenital defects in identical twins is a rarity. It is to be remembered that uniovular twins may show their similarities on corresponding sides of their bodies or on opposite sides, i.e., they may be exact copies or mirror images of one another. Furthermore it has been concluded that on the basis of studies of monozygotic twins the genes have an important bearing on the development of orthopedic as well as various other deformities, but other factors are not excluded.

The occurrence of congenital coxa vara in identical twins is not only an unusual finding but offers an opportunity to speculate on the etiology of this uncommon orthopedic deformity. Various theories regarding the etiology of coxa vara have been advanced but, as in other congenital anomalies, they are purely hypothetical. Alsberg,[1] Hoffa,[2] Kredel,[3] and Reiner[4] thought that because of intrauterine pressure the neck of the femur loses its power of growth. Bohm[5] thought it was atavism and, in support of this idea, pointed out that in anthropoid apes the femoral neck is nearly at a right angle with the shaft of the femur. Fairbanks[6] disagreed with this view and the findings. Helbing[7] thought it was due to a delay or lack of proper ossification. Nilsonne[8] suggested that the anomaly of the neck of the femur may be due to an embryonic vascular disturbance and Noble and Hauser[9] agreed with this theory.

This vascular disturbance might upset the ossification of the femoral neck and the epiphyseal line. Such disturbance may be of various gradations. If severe, there may be a complete absence of the upper end of the femur. If moderate, and this is the usual type of disturbance, the inferior angle of the neck fails to ossify or it ossifies as a separate triangular piece of bone. In either case the buttress action of the inferior angle of the femoral neck for the head has been removed, which allows the head to slip downward. This deformity usually appears after weight-bearing and is then a progressive deformity. This theory seems to offer the best explanation of the deformity and seems more practical in view of the finding of identical deformities in twins.

REPORT OF CASES

CASE 1. L. S., (Fig. 1) was a colored boy, aged eleven years, who was admitted to the New York Orthopedic Dispensary, December 28, 1933, complaining of chronic fatigue in his feet and legs. His mother stated that he walked on his toes and that his heels were always raised. He was the oldest of twins and had a normal delivery at birth. He had measles at the age of six months; otherwise no past illnesses. January 19, 1934 he was admitted to the hospital, at which time physical examination showed that the right shoulder was slightly higher than the left; there was a mild, sweeping, right thoracolumbar curvature of the spine, which disappeared on lying and the right testicle was lower than the left. The greater trochanter on the right was higher than on the left and was one inch above Nelaton's line. The right hip showed 20 degrees flexion deformity. Flexion of the femur was from 160 to 50 degrees; abduction 10 degrees; adduction 30 degrees; internal rotation 20 degrees; and external rotation 40 degrees. Muscle power was good in all groups. There was no telescoping of the femur. The right foot was in 20 degrees equinus and the left was negative. The right lower extremity was 1¼ inches shorter than the left. He walked with a slight, short-leg limp on the right and with this heel one inch off the floor.

Roentgenogram (Fig. 3) showed bilateral coxa vara, very marked on the right.

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January 29, 1934 a right subtrochanteric osteotomy was done, at which time the distal fragment was placed in 25 degrees abduction and 20 degrees flexion on the proximal. Convalescence was uneventful and he was allowed up walking in fourteen weeks postoperatively, at which time the osteotomy site was solidly united.

On follow-up examination May 2, 1936 the patient denied any symptoms; he plays all games, and recently had walked forty blocks. There was 1⅛ inches shortening of the right lower extremity. Motion in the right hip at this time was: flexion 170 to 80 degrees; abduction 15 degrees; adduction 15 degrees; internal rotation 30 degrees; external rotation 20 degrees. Roentgenograms showed union of the upper femoral epiphysis with the femoral head and neck in much better relationship with the femoral shaft.

Fig. 1. Twin (L. S.) reported in Case 1. Note appearance similar to that of Fig. 2.

Fig. 2. Twin (R. S.) reported in Case 11.

Case II. R. S., (Fig. 2) a colored boy, aged eleven years, the twin of patient 1. He was the younger of the two. He was admitted to the dispensary on the same date, had the same complaint, and had had measles at the age of six months, as had his brother, for his only past illness. He also was admitted to the hospital January 19, 1934, at which time physical examination revealed almost the same findings as in the case of his brother but on the opposite side of his body. His left shoulder was slightly higher than the right, there was a mild, sweeping, left thoracolumbar curvature of the spine, which disappeared on lying and the left testicle was lower than the right. The left greater trochanter was higher than the right and its upper tip was 1¼ inches above Nelaton's line. The joint was stable, allowing no telescoping. Motion in the right hip was normal but in the left there were the following limitations: flexion 165 to 45 degrees; abduction 20 degrees; adduction 30 degrees; internal rotation 20 degrees; external rotation 25 degrees. The left foot was in 20 degrees equinus and the right was negative. The left lower extremity was 1⅝ inches shorter than the right. He walked with a slight, left, short-leg limp, the left heel not touching the floor by 1½ inches.

Roentgenogram (Fig. 4) of the hips showed coxa vara, very marked on the left.

January 22, 1934 a left subtrochanteric osteotomy was done, at which time the distal fragment was placed in 30 degrees abduction and 20 degrees flexion on the proximal. Convalescence was uneventful and he was allowed up walking fourteen weeks postoperatively, at which time the osteotomy site was solidly united. On follow-up examination, May 2, 1934, he denied any symptoms. He runs and plays all games and keeps up with other boys. He recently walked twenty blocks without fatigue. There was 1¼ inches shortening of the left lower extremity. Motion in the left hip was: flexion 170 to 60 degrees; abduction 20 degrees; adduction 20 degrees; internal rotation 30 degrees; external rotation 30 degrees. Roentgenogram showed union of the upper femoral epiphysis on the left, with the femoral head and neck in much better relationship to the femoral shaft.

Fig. 3. Preoperative roentgenogram of twin in Figure 1; coxa vara is bilateral, being very marked on the right.

Fig. 4. Preoperative roentgenogram of twin in Figure 2; coxa vara very marked on the left.

COMMENT

The finding of coxa vara in identical twins, with the deformity in one being the mirror image in the other, seems to add support to the theory of hypoplasia. The occurrence of a deformity in identical twins would seem to indicate some hereditary constitutional factor, while the development of a deformity in one twin and not

in the other would seem to indicate some other etiological factor. Familial types of coxa vara have been described by Barrington-Ward[10] and Francke,[11] and these seem to add support to the hereditary theory.

The findings here would tend to discredit another etiological hypothesis, viz., rickets. This disease has been used to explain all bony deformities of obscure pathology. While it is conceivable that these 2 patients may have had rickets, they certainly did not show any of the characteristic features of rickets when admitted. Furthermore it is hardly probable that rickets would have produced such a mirror image deformity in these two boys. Still another time-worn theory, birth injury, may be discarded by the same reasoning.

A similarity that is as marked as in these boys, and extends alike to appearance and to similar congenital deformities, must be traceable to an inherited defective anlage.

CONCLUSIONS

The various theories regarding the etiology of congenital coxa vara are presented. The theory of embryonic vascular disturbance of the femoral neck and upper femoral epiphysis, due to germinal defect, is considered the most logical.

Two cases of congenital coxa vara occurring in identical twins, with the deformity as well as other physical features being the mirror image in the two boys, are presented.

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OS SUBCALCIS*

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MANY accessory bones of the foot have been described and several are of such common occurrence as to be quite familiar to all of us, notably the os trigonum, the os tibiale externum, and the os intermetatarseum. Dwight's classic work[1] lists these different types of accessory os calcis: the os sustentaculi, the peroneal spine, and the secondary os calcis. The first, at the hind end of the sustenaculum, is rare, having been seen only twice as a separate bone by Pfitzner[2] whom Dwight considered to "occupy a rank by himself among students of variations of hands and feet." The second is more properly an inconstant process on the lateral surface of the os calcis separating the tendons of the peroneus longus and brevis, while the more common secondary os calcis is situated either in front of the sustentaculum or in front of the processus anterior calcanei tending to articulate either with scaphoid or cuboid. A number of cases of this bone have been reported notably by Mercer[3] and it can be of clinical significance as in the case reported by Krida.[4] Nowhere can I find mention of such a bone as is reported here. The unique case reported by Heimerzheim[5] at first seems comparable, but inasmuch as his ossicle is placed far more posteriorly, resembling a normal developing apophysis at the age of ten years, (although the patient was thirty), I believe it to be different. I can agree with his conclusion that his case is the result of fragmentation of the apophysis, a rather far fetched conclusion in the following case.

A sixty-one year old farmer was seen at the Isaac Folsom Clinic, November 25, 1935, with a complaint of pain in the left heel of fourteen months duration. There seemed to have been no precipitating illness or accident except that nineteen years previously he had fallen from a light pole injuring both feet and suffering six weeks disability from which he recovered completely. His present distress began spontaneously as a sharp pain in the left heel as though a nail of his shoe were coming through. He padded the shoe and limped around with constantly increasing disability. About four months after the onset he noted a mass in the soft tissues which seemed to have been increasing in size for the past four months. Physical examination was irrelevant except locally where a mass was both visible and palpable. This was hard and slightly movable. It was tender on pressure and also on motion. There were no signs of inflammation nor any surrounding thickening.

The roentgenologist, Dr. D. A. Rhinehart, reported "a small bony structure in the sole of the left foot under the tuberosity of the os calcis. This looks like a piece of bone. There are no spurs."

A week later this ossicle was removed under local anesthesia by cutting directly down on it in the midline and shelling it out. There was a fibrous attachment to the os calcis and no cartilaginous articulation. The specimen was a rounded piece of bone about 1 cm. in diameter covered with fibrous tissue. The pathological report of Dr. A. F. DeGroat was: "Grossly the specimen consists of a smooth rounded, hard structure which when cut through appears to be a spongy bone covered by cartilage. The maximum diameter is 0.9 cm. Microscopically the bony tissue consists of well oriented trabeculae. The cortex is extremely thin and in places incomplete. It merges with a layer of hyaline cartilage. Peripherally this hyaline cartilage becomes fibrocartilage and this in turn merges with a layer of dense fibrous tissue. The cartilaginous and fibrous layer varies from 0.5 to 1.0 mm. thick."

The outcome was uneventful recovery so that when seen on August 15, 1936, he stated that he walked all day either on plowed land or city streets without any discomfort whatsoever.

In this case two questions would naturally arise. First, is this truly an accessory bone and not new growth or ossifying in-

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flammatory reaction such as we sometimes see in muscles or connective tissue? The fact that trouble did not arise for sixty sixty some slight contusion started off a chain of symptoms that would not have arisen when the factor of safety was higher.

Fig. 1. Note especially the normal trabeculation of the ossicle and the thickening of the subjacent bone. The normal foot is shown for comparison.

years of his life and that he believed the mass to have arisen ten months before being seen and to have grown during that interval lends color to that view. But many a palpable structural abnormality of the body is not noted until it becomes painful and the apparent growth could easily be imaginary. The really convincing factor however is the microscopic examination of the specimen so utterly devoid of suggestion of abnormal origin. The second question that arises, is why did such a structure if present since birth not cause trouble long before sixty? To this question it can only be suggested that in youth the adaptive faculties of the body are great and gradually decrease with age so that at

SUMMARY

A new accessory bone, the os subcalcis, is described.

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CHRONIC DISLOCATION OF ELBOW*

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L. R., aged thirty-seven years, injured her right elbow by direct trauma on July 1, 1936. Roentgenogram failed to reveal any fracture or dislocation in this region (Fig. 1).

FIG. 1. Lateral view taken on July 1, 1936, day of injury, showing negative findings.

FIG. 2. July 31, 1936, on admission; definite posterior dislocation of the radius and ulna with beginning osteoplastic formation anteriorly and posteriorly.

After sling immobilization for three days, her elbow was exercised daily by a visiting nurse until nine days after injury, when undue deformity and increasing painful disability of the right elbow became apparent. She failed to seek immediate medical attention despite marked disability of the right elbow associated with an almost fixed extension deformity of the forearm. She was seen in the Out-Patient Department four and one-half weeks following the initial trauma, when the clinical and roentgenographic pictures revealed unquestionable posterior dislocation of the right elbow (Fig. 2). At this time, she complained of slight paresthesias in the fourth and fifth fingers of the right hand. She was admitted to the hospital and an attempt at closed reduction, under fluoroscopic control, failed.

Open reduction was performed August 7, 1936, by the method of Vangorder.[3] Through a posterior midline incision, the ulnar nerve was isolated in its groove, found slightly thickened and transposed anteriorly. The triceps tendon was severed transversely ½ inch above the olecranon process, the posterior space between this tendon, the lower posterior

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humeral surface and the olecranon incisura evacuated of considerable detritus and fibrous tissue, and reduction easily effected. With the forearm at right angles, the resulting 2-inch gap in the triceps tendon was closed with a layer of transplanted fascia lata and the extremity immobilized in plaster-of-Paris bandage.

Roentgenogram the following day revealed recurrence of the posterior dislocation as well as some lateral displacement of the radius and ulna, not previously present. Correction was easily obtained under fluoroscopic control and plaster fixation reapplied. *X*-ray plates revealed reduction of the dislocation in both planes, except for undue gaping between the trochlea and ulnar semilunar notch apparent on the lateral view (Fig. 3), and this, despite thorough evacuation of the latter at the time of open reduction.

During the ensuing week, she developed early signs and symptoms of ulnar nerve compression which subsided in a few days with sinusoidal stimulation through an aperture in the plaster bandage.

Fig. 3. August 8, 1936, following the first open procedure. Reduction complete except for a marked hiatus between the trochlea and semilunar notch due to the severed triceps mechanism.

Fig. 4. October 9, 1936, complete reduction present one month after the second open procedure. The hiatus is no longer present. Note moderate osteoplastic reformation anteriorly and posteriorly.

At the end of two weeks, active exercises were initiated, revealing at first an excellent range of flexion and extension, with complete restoration of the triceps mechanism. In the ensuing days, there was gradual loss in the range of flexion. Roentgenogram one month postoperatively revealed recurrence of the posterior dislocation.

On September 9, 1936, two months after the initial injury, open correction was again performed, but through a lateral approach, since the posterior one had already proved itself unsatisfactory and the medial one, although preferable, seemed contraindicated by localized induration, redness and tenderness in the course of the transposed ulnar nerve. Considerable debris and calcific material was evacuated from the anterior and posterior compartments of the elbow joint, and reduction easily obtained without altering the integrity of the triceps tendon or the lateral ligaments, and the forearm maintained in acute flexion.

Two weeks later, active exercises were again instituted with gradually increasing range of extension from the acutely flexed position. Roentgenogram taken one month following the second open procedure revealed maintenance of the reduction (Fig. 4), with excellent alinement of the bones of the arm and forearm.

Six months following the second operation and eight months after the initial injury, the patient presents a painless, normal-appearing elbow with an excellent range of motion from complete flexion to 155 degrees of extension and pronation of 65 degrees to almost complete supination. There is no lateral instability. Except for fleeting paresthesias in the fifth finger of the right hand, there are no further evidences of ulnar nerve involvement.

DISCUSSION

This case presents numerous instructive phases.

1. The danger of over-zealous activity in a recently injured elbow, despite negative *x*-ray findings, in the light of possible soft tissue laceration and pathologic joint effusion and the danger of secondary luxation.

2. The inadvisability of attempting closed reduction in cases of three weeks duration or older, in the face of almost certain failure, and the danger of increasing ossifying periarticular changes, nerve injury and fracture.[1]

3. The inadequacy of the commonly advocated posterior approach[2–6] and the danger in the necessary severance of the triceps tendon. The latter procedure, as the above roentgenograms evidence, destroys the essential components of the Jones' position of acute flexion, i.e., the posterior sling effect of the intact triceps tendon and its coaptating force approximating the trochlea within the semilunar notch of the ulna, thus encouraging redislocation. The medial incision would serve as a more ideal approach.

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HYDROARTHROSIS OF ALLERGIC ORIGIN*

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INVOLVEMENT of the joints in allergic conditions has been observed for a number of years. However, the medical literature is very sparse in calling attention to this particular manifestation. Von Pirque and Schick very early referred to the involvement of the joints in serum disease. According to their observations, swelling was not a prominent finding. Other observers have frequently called attention to swelling of the joints as a prominent symptom as well as to pain and tenderness of the joints in serum disease.

Apparently little attention has been noted as to the importance of food as a factor in the production of intermittent hydroarthrosis. A few cases have been reported but for the most part very little is found in the literature. The fact that intermittent hydroarthrosis appears with considerable periodicity is suggestive of a possible allergic basis. Miller and Lewin[1] in 1924 treated cases by the intravenous injection of peptone solution. The response to this treatment was such as to suggest to them that idiopathic hydroarthrosis might be an allergic disease.

In going over a group of cases during the past year and a half, to determine something of the frequency of occurrence of this condition a group of 4 well defined cases were found. These 4 cases are reported as follows:

CASE I. Female, aged thirty-four years, presented a past history of hay fever at thirteen years and none since the age of fifteen. She has had frequent gastric disturbances, especially bloating, after meals for past ten years. In the family history, the mother had migraine; one son had asthma and one son had gastrointestinal allergy. The present history began at the age of thirty years with pain in the right shoulder and limitation of motion. The pain would be severe for three to five days and usually disappeared in ten days to two weeks. The attacks first came at irregular intervals. The patient finally noticed an association with the eating of chocolate. In 1934 the patient was food tested and found to react to milk, oats, apples, cherries, beans, potatoes, chocolate and feathers. The blood at this time showed a 6 per cent eosinophilia. When these foods were avoided she had relief from her gastrointestinal symptoms as well as from pain in shoulder. When the patient failed to observe the restricted foods and especially upon taking chocolate malted milks she would invariably have gastric disturbances and an exacerbation of her hydroarthrosis. She was given milk and chocolate in large quantities at one time and within twenty-four hours severe pain occurred in the shoulders. An *x*-ray picture was taken at this time but showed no evidence of arthritic change in the joints. Motion was greatly limited and the pain was severe. Examination by an orthopedist confirmed the diagnosis of hydroarthrosis. A series of blood counts showed a normal level of 6200 to 6800 and following the injestion of milk and chocolate the leucocyte count fell to 4050.

CASE II. Male, aged forty-six years gave a family history of his mother having hay fever and one sister having asthma. He has had asthma for the last three years and gastrointestinal allergy for seven years. His present illness began about three years ago shortly after the onset of an attack of asthma. He sought consultation for his asthma during the past year. During his severe paroxysms he would develop tenosynovitis and hydroarthrosis of both hands and fingers. The involvement of the hands would presist for a week or more after the asthma would subside. The hands were stiff, swollen and painful. The patient was tested for foods, pollens and epidermals and he reacted to a great many. He has been practi-

[1] MILLER, J. L. and LEWIN, PHILIP. *J. A. M. A.*, 82: 1177, 1924.

* From the Colorado Foundation for Research in Tuberculosis, Colorado College.

cally free of asthma for the past six months and has had no evidence of tenosynovitis or hydroarthrosis. In addition to the elimination of foods and epidermals he received a bacterial vaccine. On one occasion, following an overdose, he had a marked local reaction, a severe asthmatic attack followed and at the same time a hydroarthrosis and tenosynovitis of both hands developed.

Case III. Female, widow, aged fifty-nine years gave a past history of hay fever, mild in type and a gastrointestinal allergy for the past fifteen years. One son is asthmatic. Present Illness: Patient sought consultation because of persistent pain in the upper thoracic and cervical vertebral region. Also pain in both shoulders and right knee. Roentgenograms showed some lipping of the vertebra and arthritic changes in the shoulder and knee joints. Shortly after the first consultation an acute condition developed in the knee and the patient was referred to an orthopedist. A diagnosis of acute arthritis was made and the patient treated accordingly. The shoulders and cervical vertebra troubled her considerably at the same time although there was no evidence of an acute condition. After her recovery of the acute arthritis the patient returned for food tests and was found sensitive to 22 different foods. Following the removal of foods the patient had relief of symptoms in the shoulders and in the cervical region. The patient has found that the eating of any citrus fruit caused an immediate return of symptoms involving the shoulder as well as the cervical region. Because of the marked limitation of motion as well as pain, a hydroarthrosis was considered to exist in the shoulder region.

Case IV. Female, dressmaker, aged fifty-eight years, gave a history of gastrointestinal allergy for the past three years. Her present illness began as hives three months previous to consultation. Shortly after the onset of hives the patient noticed stiffness and swelling involving the joint in the hand. Very little pain was present early in this condition. About two months later both shoulders became involved. There was limitation of motion and pain on motion. The hands at this time had become so involved that it was necessary to cease work. The patient was tested for foods and failed to react. *X*-ray studies of the teeth, sinuses and gall bladder were negative. The patient was put on an elimination diet and it was found that on the elimination of wheat, beans and potatoes she was free of pain and swelling in the hands and shoulder. The hives were somewhat improved but persisted until splenic fluid was given and this has given her relief from hives. The effect of the three foods eliminated was checked by the method outlined by Vaughn. After determining the normal leucocyte level of the blood one of the foods was given and counts made at fifteen minute intervals. In each case a definite leucopenia was evident.

Three additional cases were noted in which the symptoms of a hydroarthrosis were somewhat less apparent. They are not reported as they were not as completely investigated.

DISCUSSION

The occurrence of hydroarthrosis is probably a much more common manifestation of sensitization than the medical literature would seem to indicate. Due to the fact that hydroarthrosis occurs, in most cases in association with some other allergic manifestation and is relieved by measures that cure the more severe symptoms, very little attention is devoted to it.

From the cases cited gastrointestinal symptoms were present and preceded by a considerable time the appearance of joint symptoms. It would seem that in cases of hydroarthrosis or other abnormal joint cases in which a history of food idiosyncrasy or digestive difficulty is encountered that the allergic phase should be investigated. Even in cases of arthritis, where the individual is a known allergic, it is often possible to give considerable relief by removing offending allergens. Food allergy is unquestionably a factor of great importance in hydroarthrosis, especially when it is present along with some other allergic manifestation. When this condition occurs in cases giving either a present or family history of allergic disease it would seem that allergy should be ruled out as a possible cause.

CONCLUSION

1. Four cases having allergic histories accompanied by joint symptoms are presented. The relief of symptoms by correction of the existing allergy indicates the need for the consideration of this form of treatment in cases of hydroarthrosis.

PEPTIC ULCER, RECURRENT, PERFORATING THREE TIMES WITH RECOVERY

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RECURRENT perforating peptic ulcer while quite rare is not entirely unknown. However, we have been unable to find any case reported either in domestic or foreign literature where perforation occurred three times in the same individual and upon each occasion the catastrophe came under the observation of the same physician (Sands). For this reason, the following instance appears worth recording from the standpoint of interesting medical curiosities.

Owing to the lapse of time and the imperfect hospital records which were the rule twenty years ago, the details of the first perforation are not available. However, the facts are essentially as follows:

The patient, Mr. G. H., at that time twenty-nine years of age, was operated upon at St. Catherine Hospital in 1916, for a perforated duodenal ulcer. His condition was very grave and simple closure was performed. The convalescence was quite stormy and his recovery very slow, but he was eventually restored to health. Following this happening, he dieted himself for about two years.

Again in 1925 while living in Grand Rapids, he was somewhat disturbed by epigastric pain and was treated by a physician in that city who could find no evidence of ulcer at that time.

On January 24, 1932, patient consulted Sands complaining of pain of severe character located high in the middle of the abdomen and to the right of the midline. After eating a heavy supper the previous evening, he was uncomfortable and took one dram of bicarbonate of soda with but very little relief. At that time he vomited once. He retired about 11:00 P.M. but was awakened at 2:15 A.M. with sudden, severe pain and vomited. Dr. Sands was called and at 4:15 A.M. administered morphine grain ½ plus atropine grain 1/150, after which the patient again vomited stomach contents flecked with bright red blood. At this time the pain was entirely in the upper abdomen, did not radiate to other parts, and according to the patient's statement was similar to that experienced with the perforation fifteen years before.

He was immediately removed to the hospital and operation performed at 6:06 A.M. under a general anesthetic. The operative findings were as follows: The old incision was opened and a small amount of gastric juice removed by suction. There was a perforation about 4 mm. in diameter on the anterior surface of the duodenum close to the pylorus. There were a few old adhesions around the pylorus, probably the result of a previous operation for perforating ulcer but the stomach and duodenum otherwise appeared normal. The indurated area around the perforation was about 1 cm. in diameter and the impression obtained was that of a relatively recent ulcer rather than an old chronic ulceration. The duodenum was brought up and the edges of the perforation scraped away with a knife and a pursestring of gastrointestinal suture inserted. The serosa was then brought together over the line of suture and covered with a piece of omentum. The peritoneum was closed without drainage. Following the operation, the convalescence was uneventful and the patient returned home on the thirteenth day. Shortly afterwards he returned to work and was somewhat careful about his diet for several years afterwards. He was repeatedly advised to have further surgical work done but disregarded the suggestion.

On September 25, 1936 he was awakened from sleep by severe upper abdominal pain. Dr. Mahoney was called to see him at 7:00 A.M. on this same date and upon inquiring into the history was informed that for the past two or three months, the patient had suffered a recurrence of ulcer symptoms, i.e., pain and distress when the stomach was empty. Since he ap-

peared rather comfortable at the first visit, simple measures were advised and watchful waiting suggested. At 7:45 A.M. of the same day, he was seized with uncontrollable abdominal pain and went into profound collapse. When seen fifteen minutes later there was every evidence of a ruptured viscus, i.e., slow pulse rate of 72, subnormal temperature of 97 degrees, pallid face, cold, drenching perspiration and board-like abdominal rigidity. The patient was immediately removed to the Santa Monica Hospital and operation performed at 10:00 A.M. under a general anesthetic.

The operative sheet shows the following notations:

The abdomen was opened through a 4 inch incision placed ½ inch to the right of the median line. The previous scar was avoided owing to the adhesions which might have been present. Upon opening the peritoneum there was a gush of clear mixed, gastric and peritoneal fluid. In all, 800 c.c. of this material was aspirated from under the dome of the diaphram (right) from the right paracolic gutter, the pelvis and the site of the rupture. A callous ulcer about 2.5 cm. in diameter was found just on the stomach side of the pylorus, and with a perforation 1 cm. in diameter situated 1 cm. to the left of the anterior pyloric vein. The edges of the perforation were treated with the actual cautery and the defect closed with interrupted sutures of fine black silk placed vertically so as not to narrow the pyloric opening. The stomach wall was very friable and a piece of omentum was then tacked over the line of suture. Exploration disclosed that the first and second portions of the duodenum were adherent to the liver, the gall bladder and the gastrohepatic ligament, evidently the result of previous perforations. The greater curvature of the stomach was not involved and the organ was sufficiently mobile that a posterior gastrojejunostomy was entirely feasible. However, the patient was in profound shock so it was deemed advisable to be content with simple closure. No drainage was employed. The patient left the operating table in profound shock, rallied very quickly, enjoyed an entirely uneventful convalescence and returned home on the ninth postoperative day. A small piece of tissue was removed from the edge of the perforation with the actual cautery and the report of Dr. A. A. Kosky, the pathologist, showed chronic inflammatory tissue.

The history and operative findings strongly suggest that this man suffered from the perforation at different times of three distinct and separate ulcers. While all were within an area probably about 3 cm. in diameter, the first two were apparently on the anterior surface of the first part of the duodenum while the third was definitely on the anterior surface of the stomach just to the left of the pyloric ring. The patient had used alkalis at intervals and followed an ulcer diet with considerable fidelity for twenty years.

SUMMARY

The case history of a patient is presented who had three distinct and separate peptic ulcers which perforated, the first in 1916, the second in 1932 and the last in 1936. On each occasion simple closure was performed due to the graveness of the illness and recovery ensued. All the perforations came under the observation of the same physician.

The patient had followed the usual medical management of ulcer with more or less fidelity for twenty years.

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GUMMA OF MUSCLE

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FORMERLY syphilis often went on to gumma formation. In modern times syphilis has been diagnosed in its early stages and so competently treated that gummata in any part of the body are seldom seen. Stokes says, "Gumma of the muscles are quite rare." However, it is important to consider the diagnosis of gumma under certain circumstances, even in this day and age, as the following case report may show.

In January, 1935, I saw a middle-aged white woman who complained of a tender mass in the abdomen just to the left of the midline beneath the costal margin. This mass had been first noticed only a few days before, her attention having been drawn to it by a sudden, sharp pain, severe enough to markedly interfere with her work as a house servant. She was quite sure the mass had not been present before that time. There was no history of trauma to the upper abdomen. She had been married twice and had given birth to seven children. There was no history of either miscarriages or abortions. Nor was there a history of a chancre or of secondary syphilitic manifestations.

Examination showed a slender, well developed, white woman, fifty years of age, complaining of a tender mass in the upper abdomen.

The pupils and knee jerks were normal. Examination of the abdomen showed it to be flat, the muscles well developed. About 3 cm. to the left of the midline, just beneath the costal margin there was a firm, tender mass about 4 by 2 by 2 cm., the long axis being parallel to the midline of the abdomen. This mass could not be displaced nor could it be traced to the midline.

The urine and blood count were normal. Roentgen-ray examination of the gastro-intestinal tract by both the oral and the rectal methods showed no abnormality.

The blood Wassermann was 4+ with both the noncholesterolized and cholesterolized antigens. Spinal fluid Wassermann was 1+ in both the cholesterolized and the acetone insoluble antigens.

The positive Wassermann suggested a gumma. Because gummata of muscles have become so rare, especially of the rectus muscle, and because of pain and tenderness in the mass, I felt very uncertain of this diagnosis. I was unable to rule out an epigastric hernia even though the mass could not be traced to the midline. A number of other possibilities were considered also: sarcoma of the rectus sheath, Paget's recurring fibroma, lipoma and rhabdomyoma of the rectus muscle. None of these conditions could definitely be ruled out. An intra-abdominal tumor was also considered and could not be excluded because the anterior abdominal wall was exceedingly thin. Neurogenic fibroma was a possibility which could not be entirely overlooked. Angioma was thought of but the firmness of the mass was against this diagnosis. Hematoma was also thought of but there was no history of trauma. Tuberculosis of the rectus muscle was considered but, besides being a very rare condition, no focus of tuberculosis could be found elsewhere in the body.

Epigastric hernia was deemed the most probable diagnosis, in spite of the fact that the linea alba seemed normal. I decided to operate. If the diagnosis of hernia proved wrong any of the other conditions, except a gumma, would, at all events, require surgical treatment.

Under a general anesthetic I explored the tumor and found it to arise from the fibers of the rectus muscle. It was hard, glistening, fibrous, and seemed to be infiltrating the muscle fibers. I thought that I was dealing with a Paget's recurring fibroma. I removed practically all of the tumor, repaired the defect in the muscle as well as possible and closed the wound without drainage.

Dr. McIlroy examined the tissue. His report is as follows:

"Specimen of: Rectus muscle.

"Specimen consists of a mass of tissue measuring in its fixed state 4 cm. in diameter and up to 1 cm. in thickness. In section it is com-

posed of firm pinkish-white cellular looking tissue.

"The tissue presents a bizarre appearance on histological examination. Areas of well preserved striated muscle with chronic inflammatory infiltration composed chiefly of lymphocyte-like cells and eosinophiles are seen among the bundles. Some portions of the muscle are separated by recently formed fibrocellular connective tissue. Other areas show intense chronic inflammatory infiltration. Scattered through these areas are numerous ghosts of muscle cells. Many portions of the section show necrosis resembling that seen in tuberculosis. Many giant cell systems are scattered throughout the section. These are made up of giant cells indistinguishable histologically from the typical Langhans' giant cells of tuberculosis. These tubercles also show the presence of epithelioid-like cells but the tubercle formation is not so well defined as that seen in chronic granulomatous inflammation due to the tubercle bacillus. Repeated examinations for tubercle bacilli by special staining methods are negative. Examination by Weigert's elastic tissue stain shows interruption and fraying of this tissue in the blood vessels. The lumen of many of the vessels is completely obliterated by an obliterating endarteritis. In many of the vessels the elastic tissue has completely disappeared.

"Histological Findings: Gumma of the rectus muscle."

The patient was given energetic antisyphilitic treatment. The wound healed by first intention. The tender mass did not recur and when the patient was seen one year after operation the area seemed normal except for the scar and some subcutaneous induration.

SUMMARY

A patient presented a tender, painful tumor in the left upper quadrant of the abdomen, near the midline. On exploration the tumor was found to be a gumma of the rectus muscle. It was cured by excision and antisyphilitic treatment.

RANULA AND SYPHILIS*

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A RANULA is a cyst in the sublingual region or the floor of the mouth and many theories have been published about the mechanism of its development. Most of them agree hypothetically that the ranula is a retention cyst of a sublingual salivary or mucous gland and grows by stenosis or obstruction of its excretory duct, caused by some inflammatory changes or their sequels. None of these theories, however, offer clear explanation of the pathogenesis of the ranula cyst.

From 1928 to 1932 I investigated syphilis of the mouth and jaw in the Stomatological Clinic of our University and treated 16 cases of ranula. In 14 of these cases the Wassermann reaction was positive in 10 and negative in 4. Of the 10 "Wassermann test positive" cases, 8 were proved syphilitic infections by their history or some distinct sequels of syphilitic diseases, and 2 were congenital.

Such an intimate relationship between ranula and syphilis stirred me to study the ranula cases minutely, from both a clinical and a pathological point of view.

The preliminary report on "Ranula and Syphilis" was read in the general meeting of the Japan Dermatological Society, held in Tokyo, in April, 1933. Since then, 18 cases have been treated in the Stomatological and the Surgical Clinics. Since April, 1934, I have been working in the Surgical Clinic. Therefore, 34 cases of ranula have been observed in all in the last eight years, 1928 to 1935. Wassermann tests were done in 32 cases, of which 23 or 71.9 per cent were positive.

The clinical features of our ranula cases are tabulated as follows:

HISTOLOGY

Histological studies on specimens of 27 cases were done by Dr. T. Takahashi, Clinical Assistant of the Stomatological Clinic, under my guidance, especially the wall tissue of the ranula cysts, and the surrounding sublingual salivary glands. In 27 cases, there are 7 Wassermann negative and 20 positive cases.

1. Report of 7 cases with negative Wassermann tests and no syphilitic evidence:

The main part of the wall of the cyst consists of mature connective tissue; a slight degeneration of its fibres is seen occasionally. The internal surface of the wall is covered only by connective tissue, its fibres are arranged densely and parallel to the inner side by pressure of the fluid content. Often waste substance adheres on the internal side of the fibrous wall. Many specimens have blood vessels, small gland acinus and small excretory ducts in the tissue of the cyst wall, occasionally slight infiltration of round cells. The sublingual salivary glands are mostly normal, but a few cases have round cell infiltration around the small excretory ducts.

2. Report of 20 cases with positive Wassermann tests and distinct syphilitic evidences:

The main part of the cyst wall is mature fibrous connective tissue. Some of the specimens have a ciliated epithelial layer on the internal side of their cyst walls. Most of them have no epithelial layer but they are covered with a layer of granulation tissue of definite thickness. In the granulation layer there is a proliferation of large

* Read before the Second Congress of the Pan-Pacific Surgical Association, Honolulu, Hawaii, August, 1936.

FIG. 1. Case VI. Left sublingual ranula. ♀ Seventeen years of age. Wassermann test negative.

FIG. 2. Case IX. Left sublingual ranula. ♀ Seventeen years of age. Wassermann test +++: congenital syphilis.

FIG. 3. Case X. Right sublingual ranula. ♂ Forty-four years of age, Wassermann test +++ eleven years after the syphilitic infection.

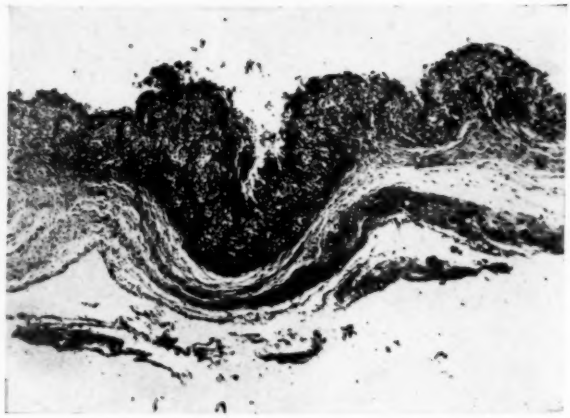

FIG. 4. Case X. The cyst wall of ranula. The internal side of the wall is covered with a layer of granulation tissue; infiltration of plasma cells.

fibroblasts, just like epitheloid cells and blood capillaries. It is infiltrated with histiocytes, round cells, and especially plasma cells. Here and there, hemorrhage and necrosis are seen. In the connective tissue, near the inner epithelial or granulation layer, there are perivascular infiltrations of plasma cells, and thickening of the intima of the blood vessels.

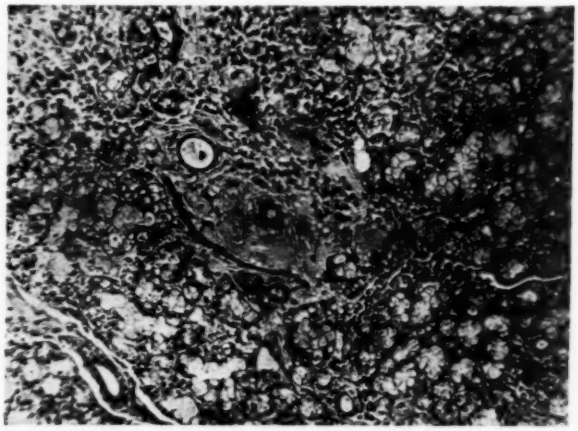

Fig. 5. Case IV. The sublingual salivary gland; chronic interstitial inflammation; infiltration of plasma cells and histiocytes; atrophy of the parenchyma cells; necrobiosis.

The sublingual salivary glands of all 20 cases show more or less a condition of chronic interstitial inflammation, i.e., distinct infiltration of round cells, especially of plasma cells, between the gland cells and around the excretory ducts. According to the grade of cell infiltration, there is atrophy of the parenchyma cells of the glands. Some of the cases have degenerative or necrotic areas with proliferation of histiocytes.

These histological features are clearly syphilitic. The syphilitic changes of sublingual salivary glands are worthy of attention.

From these findings, I would state that ⅔ of our ranula cases are distinctly syphilitic. The syphilitic changes of the sublingual salivary glands, or around their excretory ducts, are the important factor in the forming of the ranula cyst.

It has been generally experienced heretofore that the ranula cysts are divided clinically into two types:

1. Relatively smaller cysts, with thick walls, not easily ruptured at the time of removal, so that they are easily extirpated and there is usually no recurrence of this type; and

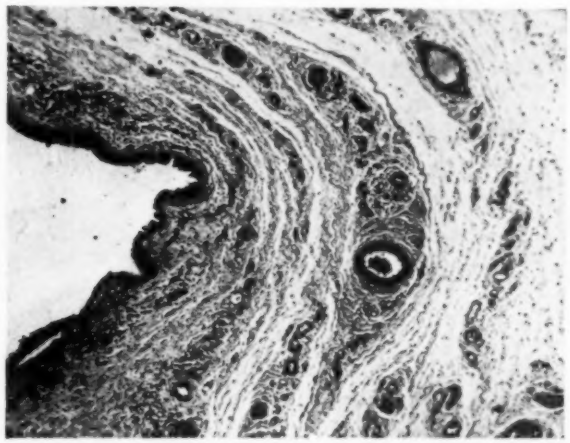

Fig. 6. Case XVI. The cyst wall of ranula; the internal side of the wall is covered with a ciliated epithelial layer; peri-vascular infiltration of plasma cells.

2. Larger cysts that grow rapidly in the tongue muscle or the floor of the mouth. Their walls are very thin and easily ruptured, so that they are difficult to extirpate

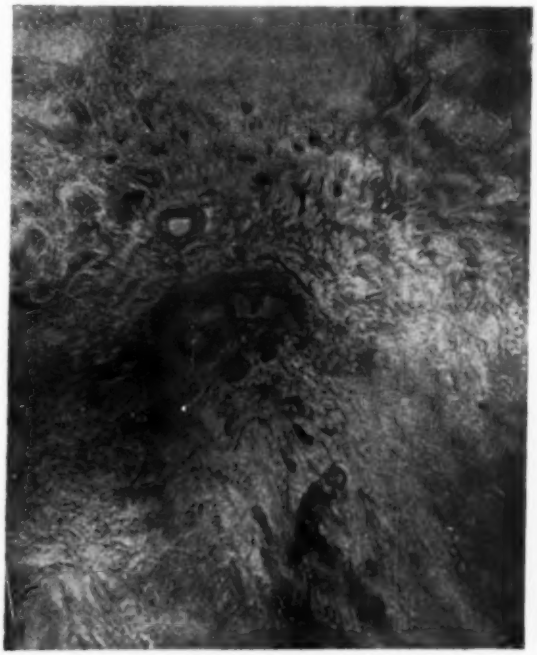

Fig. 7. Case XVI. The cyst wall of the ranula; spirochete around the small blood vessel, stained by Levaditi's method.

completely without rupture. Therefore there is frequent recurrence of this type.

Our recent clinical experience teaches us that the smaller, thick walled ranulas are usually non-syphilitic; the larger, thin walled ones are mostly syphilitic.

Since 1933 we have treated the syphilitic ranula cases in the following manner: A small anterior piece of the cyst wall in the sublingual region is cut off, large enough to drain the fluid content and then the patients are treated by antisyphilitic methods (potassium iodide, mercury, bismuth, salvarsan, etc., respectively). The cysts gradually become smaller and heal in one to two weeks. Complete cure of the patients' syphilitic condition will, of course, occur later. Occasionally we have seen some cases relapsed after the antisyphilitic treatment and drainage. Such relapsed cysts again disappear rapidly by the antisyphilitic treatment. Therefore the treatment of the large ranula cysts is greatly simplified when they are syphilitic.

PATHOGENESIS

Moreover Dr. Takahashi has performed the following research work in regard to the pathogenesis of ranula cysts.

1. The spirochetes around the small blood vessels in the cyst wall could be stained by Levaditi's method.

2. After patient effort he succeeded in inoculating the normal testis of rabbits with the wall tissue of the ranula cyst; pieces of the wall tissue from 9 cases were inoculated into 28 rabbit testis with 3 positive results. Inoculation of the fluid contents of the cyst into 16 rabbits' testis were all negative.

3. He systematically investigated 26 human specimens of sublingual Warton's duct (22 normal and 4 ranula cases) with the surrounding gland tissue, in serial sections, approximately 2500 sections for each specimen. Some deformed branch ducts were found, which are supposed to be

Case No.	Sex	Age	Duration of Disease	Location	Size	Wassermann Test	Syphilis		Treatment
							History	Sequels	
1	♀	33	6 years	left sublingual	index finger tip	.....	..	..	extirpation
2	♂	31	1 month	left sublingual	pigeon egg	−	−	−	extirpation
3	♂	5	6 months	left sublingual	thumb tip	.....	..	..	incision
4	♂	32	2 weeks	left sublingual	chicken egg	+++	+	+	extirpation
5	♂	29	1 month	right sublingual	chicken egg	+++	+	+	extirpation
6	♀	17	2 months	left sublingual	chicken egg	−	−	−	extirpation
7	♂	8	4 months	left sublingual	thumb tip	−	−	−	extirpation
8	♂	3	2 weeks	left sublingual	pigeon egg	++	congenital		incision
9	♀	17	1 month	left sublingual	chicken egg	+++	congenital		extirpation, antisyphilitic
10	♂	44	3 years	right sublingual	goose egg	+++	+	+	extirpation
11	♂	21	2 weeks	right sublingual	pigeon egg	−	−	−	extirpation
12	♀	24	1½ months	right sublingual	pigeon egg	+++	+	+	extirpation, antisyphilitic
13	♀	29	1 y. 3 m.	left sublingual	thumb tip	++	+	+	extirpation, antisyphilitic
14	♀	23	3 months	left sublingual	index finger tip	++	+	+	extirpation, antisyphilitic
15	♂	34	2 months	right sublingual	chicken egg	+++	+	+	extirpation, antisyphilitic
16	♂	33	2½ years	right sublingual	thumb tip	+++	+	+	extirpation, antisyphilitic
17	♂	31	7 months	right sublingual	pigeon egg	+++	+	+	drainage, antisyphilitic
18	♀	49	2 months	right sublingual	pigeon egg	+++	+	+	drainage, antisyphilitic
19	♀	32	1 month	left sublingual	little finger tip	−	−	−	extirpation
20	♀	48	1 month	right sublingual	pigeon egg	+++	−	−	drainage, antisyphilitic
21	♂	27	2 weeks	left sublingual	thumb tip	+++	+	+	drainage, antisyphilitic
22	♀	28	1 month	left sublingual	pigeon egg	+++	−	+	drainage, antisyphilitic
23	♂	51	8 months	left sublingual	little finger tip	+++	+	+	extirpation, antisyphilitic
24	♀	28	9 months	right sublingual	index finger tip	+++	+	+	drainage
25	♂	13	1 month	right sublingual	thumb tip	−	−	−	extirpation
26	♂	42	1 year	right sublingual	index finger tip	+++	+	+	drainage, antisyphilitic
27	♀	23	1 y. 2 m.	left sublingual	thumb tip	+++	−	+	drainage, antisyphilitic
28	♂	18	4 months	left sublingual	pigeon egg	+++	congenital		drainage, antisyphilitic
29	♀	31	1½ months	right sublingual	little finger tip	−	−	−	extirpation
30	♀	10	2 months	submental	chicken egg	−	−	−	extirpation
31	♀	13	2½ years	submental	chicken egg	+++	congenital		incision, antisyphilitic
32	♀	35	1 year	right sublingual	pigeon egg	+++	−	+	drainage, antisyphilitic
33	♂	17	1 year	right sublingual	thumb tip	−	−	−	incision
34	♀	38	5 months	left sublingual	pigeon egg	+++	+	+	drainage, antisyphilitic

one of the etiological factors for the development of a retention cyst.

CONCLUSION

1. About 70 per cent of our ranula cases are syphilitic, i.e., 23 cases of 32 are proved positive by the Wassermann test.

2. Histologically the syphilitic changes in the cyst wall and the surrounding sublingual glands are seen. Inoculation tests to rabbit testis from cyst walls are positive.

3. Syphilitic ranula cysts can be easily cured by simple drainage of the cysts and administration of antisyphilitic treatment.

The Stomatological Clinic is now in charge of Dr. T. Kanamori and I would like to express here my hearty thanks for his cooperation in referring the recent cases of the Stomatological Clinic given in this report.

INVOLVEMENT of the hip constitutes about 30 per cent of all bone and joint tuberculosis. It is more common in early childhood, 88 per cent of all cases occurring in children under ten years of age and 45 per cent between the ages of three and five years.

From—"Handbook of Orthopaedic Surgery" by Alfred Rives Shands, M.D.

NEW INSTRUMENTS

SELF-LUBRICATING CATHETERIZING FORCEPS*

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CATHETERIZATION is a common procedure carried out not only by urologists but by members of the profession at large, by the nursing profession and in certain individual instances by patients themselves.

Therefore, a procedure which is so common, and so universally used, any new improvement in technique, by virtue of a new device or instrument that gives greater protection to the patient and at the same time provides greater convenience for the physician should find a place in the armamentarium of those individuals called upon to perform this particular minor operation.

In the development of this instrument three factors were constantly kept in mind: (1) a sterile technique, (2) a thoroughly and completely lubricated catheter, and (3) the factor of convenience.

Sterile Technique. In demonstrating the steps of the procedure with this instrument you will immediately notice there is a break in absolutely sterile technique. There is a small area of contamination on the catheter, where it is gripped between the thumb and index finger of the left hand. But it so happens that this small area of contamination is so situated on the catheter that it does not enter the human body nor does it in any way contaminate the distal or syringe end of the catheter, in the event that an irrigation or instillation is to be carried out, therefore it becomes a negligible factor.

Lubricated Catheter. The lubricating property of this instrument is thought to be a very definite improvement over the older and less thorough methods. The catheter is lubricated thoroughly and completely the mechanics being such that it is almost impossible for any portion of the periphery of the catheter to escape contact with the sterile lubricant, in the collapsible, wedge shaped lubricating compartment through which it passes. The lubricant is intermittently agitated by the opening and closing of the forceps in the feeding process of the catheter thereby forcing it into contact with the catheter.

Factor of Convenience. In this era of speed, progress and advance, any new instrument or device must fulfill this requirement. Here again it is felt that this instrument definitely fulfills this requisite, in that it does away with the necessity for the use of rubber gloves in carrying out this procedure. It is somewhat of a radical deviation from the present accepted methods, it is true, but nevertheless, as the steps of the procedure are carried through with the instrument, it will be readily seen how it is possible to carry out a technique which fulfills the requirements of an aseptic or sterile technique for this particular procedure.

In demonstrating this new self-lubricating catheterizing forceps I shall go through the steps of the procedure with the instrument, a catheter, and a tube of sterile K-Y jelly.

The instrument is removed from the sterilizer and gripped in the right hand in a manner shown in Figure 4. Some sterile K-Y jelly is then expressed into the funnel-like, wedge shaped collapsible compart-

* Read before the April, 1936, meeting of the Section of Genito-urinary Surgery of the New York Academy of Medicine.

ment of the instrument. A sterile catheter is gripped between the thumb and index finger of the left hand approximately 1 to 2 inches from the distal or syringe end of the catheter. The catheter is brought into contact with the undersurface of the broad flange of the instrument as illustrated by Figure 1 and is gradually raised until the tip of the catheter automatically slips through a notch in the side wall of the lubricant compartment engaging the tip end of the catheter. Without changing one's grip on the catheter but with a slight rotary motion of it, the catheter is permitted to pass down through the lubricant compartment (Fig. 2) and out the smaller end for a distance of from 3 to 4 inches as shown in Figure 3. The end of the catheter then is looped around and laid in the side trough of the instrument as also illustrated in Figure 3. The catheter is then bent acutely back upon itself and the end

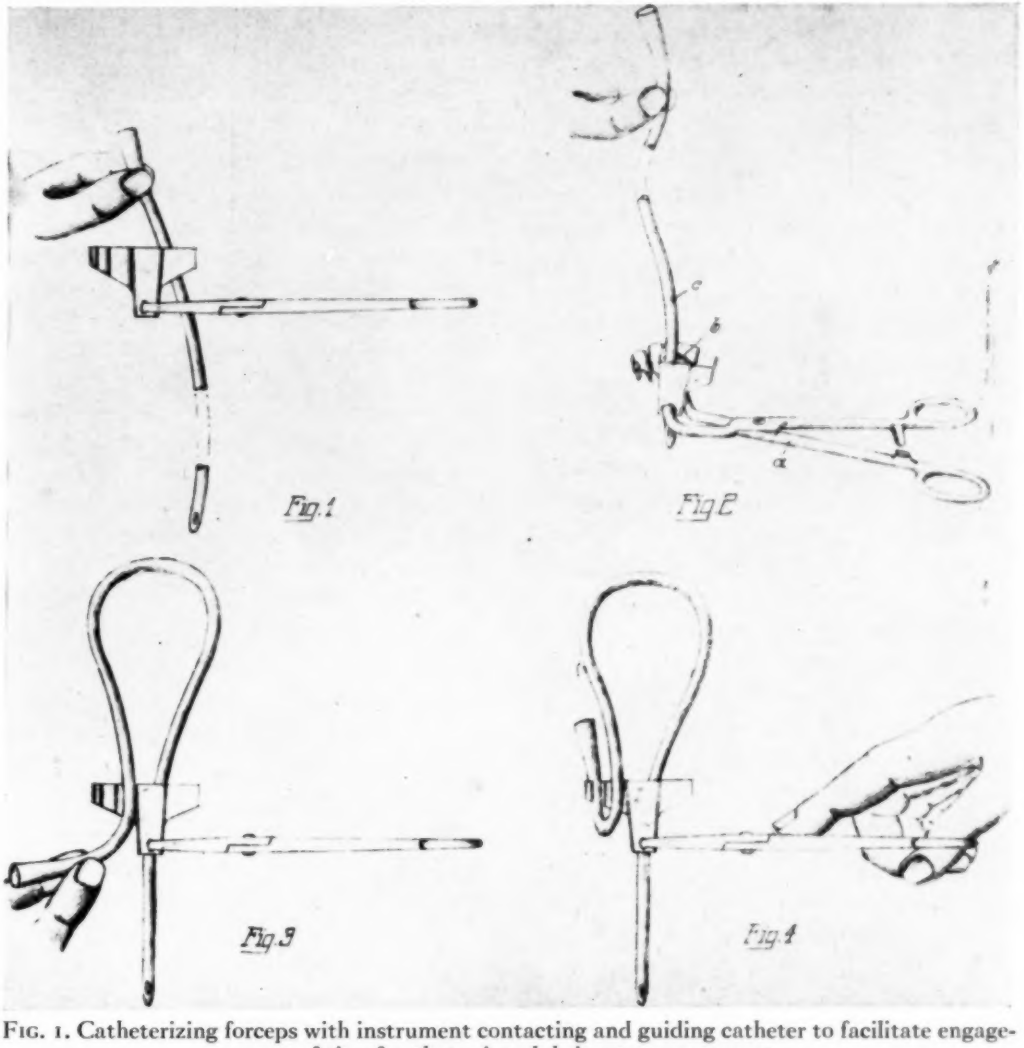

Fig. 1. Catheterizing forceps with instrument contacting and guiding catheter to facilitate engagement of tip of catheter into lubricant compartment.

Fig. 2. Catheterizing forceps with tip of catheter engaged and just emerging from small end of collapsible lubricant compartment. (a) Forceps in released position. (b) Sterile lubricant. (c) Catheter.

Fig. 3. Catheterizing forceps with three to four inches of lubricated catheter protruding from small end of instrument. Balance of the catheter is looped around, and the end laid into trough arrangement, just prior to the final anchoring of the syringe end of catheter to the instrument.

Fig. 4. Catheterizing forceps with sterile catheter in place, ready for insertion.

fastened in a hook-like arrangement on the side of the instrument, lateral to the trough. Figure 4 illustrates the catheter in place, in the instrument ready to be inserted, with the left hand free.

After the catheter enters the bladder the forceps is opened and with a slight twist of the instrument the catheter tends to straighten out, but with the syringe end of the catheter being retained in the hook on the side of the instrument which serves to control the draining end of the catheter.

The flange previously mentioned, to guide the catheter into the lubricant compartment has the added function of serving as an instrument rest, or hook to be engaged over the edge of the drain pan.

In conclusion I wish to again emphasize that the procedure of catheterization by means of this instrument may be carried out with perfectly sterile technique, without the use of rubber gloves, and a catheter passed which is thoroughly and completely lubricated. Both of these factors are very essential requisites to the procedure according to the best accepted principles of catheterization.

DISCUSSION

DR. STANLEY R. WOODRUFF: This instrument is of considerable value for two reasons: (1) It is a great thing for those patients who still catheterize themselves, because they will not wash their hands although usually we can get them to sterilize a catheter, and if they have an instrument of this description, they will sterilize it and the catheter, and will pour some lubricating jelly into the receptacle, thus making a fairly sterile instrument of the catheter. (2) This instrument will be of use in the office, particularly to those urologists who do not wear gloves in the office or change them for each patient.

SEPSIS is the greatest single cause of death in the child-bearing woman, outnumbering all other causes, and accounting in its own right for over one-fourth of all obstetric fatalities.

From—"A Textbook of Obstetrics" by Edward A. Schumann, M.D.

A MECHANICAL BREAST PUMP

CHARLES ED. WHITE, M.D.

MUSKOGEE, OKLAHOMA

THE development of the electric breast pump has been a great aid to the mother and baby. The initial cost of this instrument has made it almost prohibitive except in the larger hospitals. The rubber bulb suction type of breast pump has always been unsatisfactory. Feeling that there was a definite need for a mechanically operated breast pump which would be inexpensive, compact in size and one that could be used by patients in their homes where an electric pump would not be available, I have designed the pump described.*

As with the electric breast pump this pump has been designed to imitate the nursing baby by suction on the nipple. It can be used in emptying the breast after nursing to prevent and relieve engorged and caked breasts. It withdraws milk painlessly through fissured nipples, and may also be used to draw out flat or inverted nipples. It has been used successfully in expressing milk from the breast for premature infants.

Figure 1 shows the pump assembled. It is 8 inches in length and weighs twelve ounces. May be easily carried in a grip. By changing the position of the crank on the pump with that of the milk reservoir it may be used with either the right or the left hand. The glass shield is applied to the breast so that the nipple is in the midline with the opening of the shield.

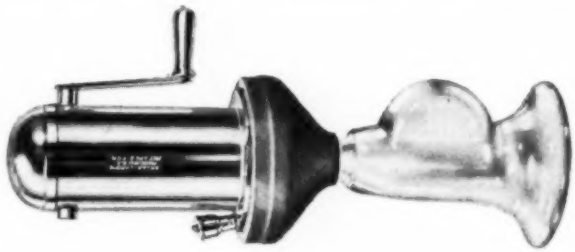

FIG. 1.

The patient or nurse governs the amount of suction applied by the rapidity in turning the crank.

The amount of suction applied to the breast is gauged by each individual patient. By touching the trigger valve with the thumb, suction is instantaneously released, giving a pulsating effect. Far more suction is produced by this breast pump than can be developed by a nursing infant.

When the milk reservoir of the breast shield is two-thirds full it is emptied by pouring the milk through the opening of the shield. The shield is then wiped off with gauze moistened in boric solution.

The pump may be separated into three parts for cleansing. The glass shield should be boiled before reusing

There is very little danger of traumatizing the breast since each patient regulates the suction necessary for her own need.

* Manufactured by J. Sklar Mfg. Co., Brooklyn, New York.

A METHOD FOR PRODUCING PYROGEN-FREE WATER FOR INTRAVENOUS THERAPY*

LEO F. BLEYER, M.D. AND M. ROHDE

ELMIRA, N. Y.

MUCH confusion still exists among clinicians about the causes of the hyperpyrexia reaction following intravenous therapy. Apparently little attention has been paid to the elaborate experimental study by Banks.[1] According to this investigator, the chief cause for the occurrence of fever and chill after venoclysis lies in the presence of substances derived from the metabolism of chromogenic bacteria which are abundantly found in river, canal, and creek water. The pyrogenic substances are characterized by extreme resistance to heat and cannot be broken down by the routine distillation process.

In our own community, filtered and chlorinated water from the Chemung river is used for drinking purposes and was also used in our hospital for the preparation of distilled water from which solutions for intravenous saline and glucose therapy were made. A startling high percentage of reactions followed the use of this water. Adjustment of the various factors previously held responsible for the hyperpyrexia reaction as *p*H range of the solutions, rubber tubing, temperature of the solutions, and speed of injection failed to bring any improvement. The fever-chill reactions persisted and forced us to approach the problem from a different angle. We became convinced that the quality of the raw water was unsuitable and that routine distillation could not destroy the substances which produce hyperpyrexia. We found samples from raw water as well as from simple and triple distillates of this water positive for pyrogen as examined by the postassium permanganate reduction test. Accepting the experimentally confirmed theory according to which pyrogenic substances are broken down by high pressure steam carried at 60 pounds in the main, we decided to work out our own mechanical device by which condensed high pressure steam could be employed for distillation instead of city water. Inquiring first about commercial stills constructed for the use of steam, two manufacturing companies informed us that they had discontinued production of such a still for the reason that it was found extremely difficult to remove the impurities such as boiler compound, grease, scale, etc., which came along with the steam from the boiler and were delivered to the evaporator of the still. This information was anything but encouraging to us. Nevertheless, we saw in the employment of condensed high pressure steam as feed water the only remedy for the removal of pyrogen. As a result, we were forced to construct an apparatus from which satisfactory feed water for redistillation can be collected.

Our system embodies the following modifications: (1) We separated the steam condensation unit entirely from the redistillation outfit so that the steam under no circumstances could be blown over into the condenser of the redistillation set and delivered as distilled water. Steam taken from the main was circulated through a large water cooled condenser of block tin from which it returned completely liquefied and was transferred by gravity flow into the feed-water inlet of the redistillation set. (2) Any scale containing condensed water which always accumulates in pipe lines when not in continuous operation had to be removed first in order to secure a stream of dry pure steam. This purification was

[1] BANKS, HORACE M. *Am. Jour. Clin. Path.*, 4: 260, 1934.

*From the Pathological and Engineering Departments of St. Joseph's Hospital, Elmira, N. Y.

accomplished by passing steam from the main at 90 to 120 pounds through a reducing valve through which the pressure was decreased to 30 to 40 pounds. A T-type pipe sector, attached with the horizontal arm to the reducing valve, was connected with the lower segment of the vertical arm through an extension line to a Webster syphon trap and with the low pressure steam return-system of the heating plant, operated continuously day and night at a 9 inch vacuum by an electrically driven pump in the boiler room. The continuous suction thus prevents any accumulation of dirt water in the pipe line and is, in addition, assisted by gravity flow. (3) Steam passing upward in the T-shaped sector to the condenser is forced through a filter specially built by one of us (M. R.) to remove any residual impurities. It consists of a wide tube of block tin filled with several layers of glass-wool and discs of glass cloth.* This filter is built into the steam pipe by screw attachment and can be easily removed for cleaning and refilling.

With the help of these three mechanical devices, we obtain a steam distillate which is perfectly clear, odorless and free of impurities, as far as can be determined by microscopical examination, centrifuging and filtration through filter paper. Upon repeated checks, the distillate was found to be entirely free of pyrogen and has a *p*H of 6. to 6.5. All glassware and rubber tubing used for intravenous therapy are rinsed exclusively with pyrogen-free steam distillate. Any contact of the inside wall with tap water is avoided. Following the use of this technique, many venoclyses have been given without reaction.

* Manufactured by Corning Glass Works.

SUMMARY

A method is briefly described by which high pressure steam can be freed from impurities in the boiler system by means of continuous vacuum scale drainage and glass wool filtration. The pyrogen-free clear steam distillate thus obtained can be advantageously employed as feed water for redistillation. The method is inexpensive and recommended to hospitals otherwise depending on pyrogen-bearing raw water.

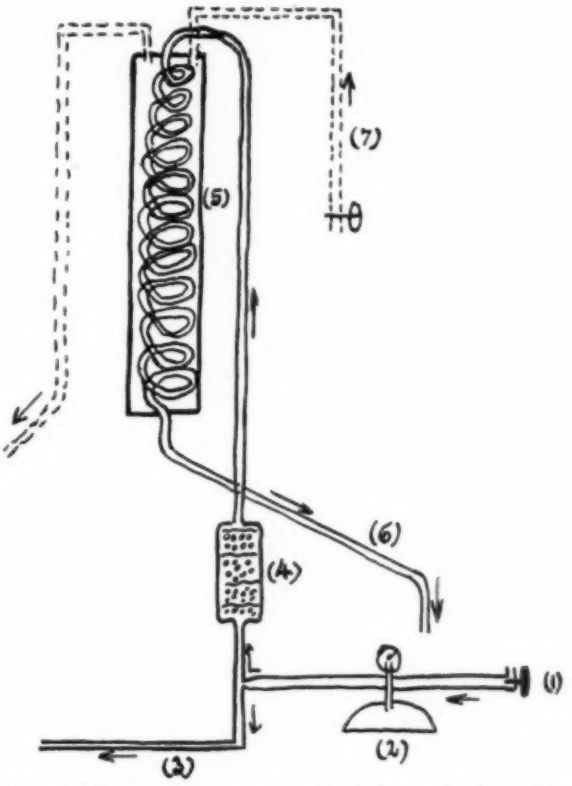

Fig. 1. Diagram of steam-condensation unit. (1) High pressure steam inlet. (2) Reducing valve. (3) Continuous vacuum scale drainage. (4) Glass wool filter. (5) Block tin condenser. (6) Condensed steam flowing into inlet of redistillation set. (7) Cooling system for condenser.

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

JOHN HUNTER*

N. P. RATHBUN, M.D.

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BROOKLYN, N. Y.

ONE approaches any consideration of the life and work of John Hunter with a certain amount of reverence and awe. From time to time, at rare intervals there stalks across the pages of history some colossal figure whose intellectual attainments seem to place him somewhat beyond the pale of ordinary mortals. I think perhaps most of us would place at the head of such a group Leonardo Da Vinci. We would probably include Goethe, perhaps Benjamin Franklin and a very few others. I do not wish to imply that even John Hunter quite measured up to the intellectual stature of Da Vinci; with one notable exception, probably no man ever did; but I do wish to imply that Hunter belonged in such a group.

On the occasion of one of the Hunterian Orations, Samuel D. Gross said of him, "With the exception of Hippocrates, he was the grandest figure in the history of medicine." Personally, I feel that Dr. Gross might safely have omitted the exception. Hunter was the greatest surgeon of his day, perhaps in point of time the first scientific surgeon of all time, as we today understand scientific surgery, he was not only the first but far and away the greatest comparative anatomist of all time. He was a great physiologist, a great zoologist, a great naturalist, a great thinker, a great man. His accomplishments in any one of the sciences which I have mentioned would have been enough to guarantee him enduring fame. He was equally great in all.

If we reflect for a moment upon the volume of work which he did over a period of forty years and that during the last twenty-five of those years he was tortured by a disease which eventually caused his death, we are bound to marvel that any ordinary mortal could do so much and we are forced to the conclusion that he was no ordinary mortal.

The time allotted to my presentation will of course permit of only the sketch of such a life and I shall therefore endeavor to give you only a few of the high lights. He was born February 14, 1728, the tenth child in a poor but genteel family living in a small town just outside of Glasgow. Several of

* Read before a meeting of the Brooklyn Hospital Staff, January, 1935.

his brothers and sisters died in early childhood. Those who survived were all regarded as particularly bright children who did well at school. One of them, William, ten years his senior, later achieved great distinction as a surgeon and anatomist in London. John from the first, was the odd Dick of the family and caused his elderly parents no end of anxiety because of his habits, his ill chosen companions and his unwillingness to take his schooling seriously. His marks were poor and his parents often discovered that many days when he was supposed to be in school had been spent by John wandering idly about the fields and woods. The neighbors thought and said "That red headed Hunter boy is a bad lot; he'll never amount to anything." There was, however, even at this time an inkling of what the man would be. While he took no interest in books, he was intensely interested in everything else about him and was constantly asking "fool" questions which no one could answer. "What makes the wind blow? Why do clouds form? Why do cuckoos lay their eggs in other birds nests?" He continued to drift along in this way until he was twenty, at which time he had accomplished exactly nothing and with apparently no prospects ahead. His parents by now, more than ever discouraged, decided upon another plan: they arranged with their eldest son, William, who was already established as a successful surgeon in London, to take his young brother into his home and see what he could do with the erring child. So John packed his bag and went off to London.

William at this time was not only a successful surgeon but as so many surgeons of those days did, he conducted a class in anatomy and one of John's first jobs on arriving in London, was to dissect a forearm in preparation for one of his brother's lectures. Here at last he found work to his liking. He did the dissection eagerly and well, quite surprising William with his skill. Many other dissections followed, all of them done with meticulous care. He did not, however, at once abandon all the bad habits of his youth. He spent much of his time in the cheapest kind of dance halls and variety theatres and associated with a gang of rowdies, all of which seemed quite out of character for the brother of the elegant and refined William Hunter, leading surgeon of a great metropolis. As time went on, his dissections consumed more and more of his time and his increasing knowledge of anatomy quite naturally led to a desire to learn something of surgery, so he registered with the classes of Cheselden and Percival Potts. His associations with these men and his brother provided him with his first knowledge of surgery.

When he was twenty-five, William, feeling that a future surgeon should acquire some educational polish, persuaded him to register for some of the courses at Oxford. This did not interest him in the least and he left at the end of two months with the feeling that these courses had not the slightest bearing on the subjects in which he was most interested. He said himself of his Oxford course, "They tried to make an old woman of me; they wanted to stuff me with Greek and Latin but these schemes I cracked like so many vermin as they came before me." His utter lack of educational attainments outside of his own chosen fields was apparent throughout his life. He published nothing until after he was forty and even then all of his papers and books required a great deal of editing by his associates for the purpose of correcting the most glaring errors in spelling and grammar. He was a poor talker and his early lectures, begun in his brother's school at the age of twenty-six, were always read from manuscript. They were put together badly and poorly delivered and yet in spite of this fact they were so replete with original thought and observation that he came eventually to have larger classes than any of his fellow surgeons and he attracted students from all over the world, many of them men who later became famous. To mention a few, there were Sir Astly Cooper, Phyne the father of surgery in America, and most famous of all, Edward Jenner.

His association with Jenner as a pupil was the beginning of what proved to be a life-long friendship and his letters to Jenner provide an interesting sidelight on the lives of both of these great men.

His contemporaries often commented upon and criticized his lack of refinement and education. Hunter once said of one of them "Jesse Foote accuses me of not understanding the dead languages; but I could teach him on the dead body what he never knew in any language, living or dead."

In 1760 he enlisted as an army surgeon and was stationed at Belleisle. He remained in the service for two years and during this time, of course, acquired a deal of experience in gunshot wounds and other forms of traumatic surgery. To illustrate the type of experience and also to illustrate his literary style of those days, I quote from a letter to his brother. He was describing a case that he was called upon to treat and he said, "the ball went into the man's belly and hit his guts such a damned thump that they mortified."

On leaving the service he returned to London, established a lecture course of his own, opened an office for the practice of surgery and secured an appointment on the surgical staff of St. George's Hospital and now began a thirty year period of the most amazing productive activity that the world has ever seen in any line of endeavor. He worked on his experiments and his dissections every day from early morning until late at night, with hardly ever an intermission. He even begrudged the time spent in earning a living. He once said to one of his laboratory assistants, "I suppose I must go and earn this damn guinea or I shall be wanting it tomorrow."

His dissections included almost every known member of the vegetable and animal kingdom: insects, reptiles, fish, even a whale.

He very early conceived the idea of creating a museum which would illustrate all phases of the structure and function of all forms of animal and vegetable life. This collection grew rapidly during those intensely active years and is now in possession of The Royal College of Surgeons and constitutes today the greatest collection of its sort in the world.

When he had earned enough money from his surgical practice (and he spent most of the money that he made throughout his life on his museum) he bought a country place just outside of London and this he populated with the weirdest collection of animals that were ever gotten together either before or since: all of the so-called domestic variety, frogs, snakes and lizards, eagles and cuckoos, leopards and deer. He was on the most intimate terms with all of them, studying their life history, their habits, their body temperature, their means of propagation, their development during life and their structure, including the finest details, after death. He is said to have carefully examined every individual member of more than a dozen hives of bees in order to prove that following the impregnation of the Queen bee no males remain in the hive.

He was constantly writing to Jenner asking him to send along any other animals that he did not have in his collection. If he could not send the animals, then try certain experiments: determine the temperature of the hedgehog during hibernation, check up on the viscosity of the blood during this period. It might be interesting at this time to mention the fact that Hunter throughout his life played with the idea of securing some animal, preferably a human one, imbedding him in ice to be left or kept in this state for a period of a hundred years, at which time he hoped he might be resurrected in perfect health.

On one occasion he tied the external carotid of a young deer and noted, as he expected to do, that the antler on the corresponding side was cold. Ten days later he noted much to his surprise that the antler was again warm and was growing. He suspected the reason but as was true of all of his experiments, he took nothing for granted. The deer was sacrificed and the collateral circulation demonstrated. Not

long after a man was admitted to his ward at the hospital suffering from a popliteal aneurism. Such cases had in the past been treated by amputation. Remembering the lesson of the deer he exposed and ligated the femoral artery, in what has since been known as Hunter's canal. A few weeks later the patient walked out of the hospital cured and with a good leg. The principle involved of course constitutes one of the great contributions to surgery.

During the early part of this period he made copious notes but published little or nothing. In 1767 he was elected a member of the Royal Society and following this he began making frequent contributions to the literature, which he continued to do throughout the remainder of his lifetime. His published works, including his notes on his dissections and experiments, but not including a great mass of manuscript which was never published and much of which was destroyed by fire after his death, comprises many volumes. To illustrate the wide variety of his activities I will mention just a few of the titles: "Observations on the Gillaroo Trout," "Experiments on animals and vegetables with respect to the power of producing heat," "Account of the Organ of Hearing in Fishes," "An experiment to determine the effect of extirpating one ovary upon the number of young produced," "Observations upon the structure and economy of whales," "A treatise on the venereal disease," "A treatise on the natural history of the human teeth," "A treatise on the blood," etc., etc.

In May 1771, at the age of forty-three, he married a Miss Anne Home. There is every reason to believe that they were congenial and yet I am sure that Mrs. Hunter led the sort of life which wives of geniuses are all supposed to live. He had an irascible temper and in fact died in a fit of temper. The story is told that on one occasion he returned to his home and found Mrs. Hunter entertaining a large group of notables. He blustered into the reception room, announced in a loud voice that he had no idea what brought all these people to his home, but in any event that he had work to do, that he did not propose to be annoyed and ordered them to get out at once. History does not relate what Mrs. Hunter said to him after the guests departed.

As a clinical surgeon, Hunter was eminently successful and following the death of Percival Potts, who was for a time his only rival, he was for the last five years of his life easily the one outstanding surgeon of all England or perhaps the world.

Notwithstanding the fact that he begrudged every moment of time spent away from his laboratory, it is evident that he was painstakingly careful in his clinical work. He said of one patient, whose case he was reporting, "After talking to this man for over an hour and then making a thorough examination, I was able to elicit one or two facts that appeared to have a bearing on his problem."

During his latter years he was very successful from a financial point of view, his income rising to five thousand pounds annually, which in those days represented wealth "beyond the dreams of avarice." However, he saved little or nothing from this large income. Not only did he maintain an expensive home with many servants but every spare shilling went to the rapidly growing museum and its upkeep.

During the last twenty years of his life Hunter suffered from frequent attacks of angina. There is nothing to indicate that he understood the pathology of this disease but he was thoroughly familiar with all its clinical phenomena and its inevitable end. He said one time, "My life is always at the mercy of any scoundrel who may choose to annoy me." This comment was, in a sense, prophetic. In the autumn of 1793, at the age of sixty-five, while attending a medical board meeting he became enraged at some remark made by another member of the board, attempted to speak, gasped, collapsed in his chair and was carried to an adjoining room, where he died a few moments later. He was buried at St. Martins-in-the-Fields. Sixty-six years later

in 1859 his body was removed to Westminster Abbey, where it now rests with many of Britain's other great dead.

His principal asset, in fact almost his only asset at the time of his death was his museum. This was of course totally unproductive from a monetary point of view so that his family were poorly provided for. Parliament made a few small money grants and it was urged upon Mr. Pitt, the prime minister, that the Government should buy the collection. He responded that he could not see any reason for spending money on specimens when there was hardly enough money in the treasury to pay the gunpowder bills. Posterity owes much to Everard Home, a brother-in-law of Hunter's and to William Clift, one of his laboratory assistants for their efforts in keeping the collection intact for the next six years. In 1799 Parliament came to understand the importance of preserving this magnificent collection for future generations and purchased it for the sum of fifteen thousand pounds. It was placed in charge of The Royal Society and later the Royal College of Surgeons, where it may be seen today in their quarters at Lincoln Inn Fields and constituting even today the greatest collection of its sort in the world. It consists of 13,682 specimens, carefully prepared and mounted with Hunter's own hands and ranging in variety all the way from the tiniest insect through pathological specimens and the skulls of all nations to the skeleton of the Irish Giant.

William Clift, after a careful survey of the museum and the manuscripts published and unpublished, estimated that Hunter had dissected, in addition to hundreds of human bodies, well over 500 different species of animals, including insects, reptiles, fish, birds and mammals and that in many instances there had been many dissections of different specimens of the same species. Copious notes and often drawings were made for each dissection.

Hunter left in addition to his museum and published writings many volumes of unpublished manuscripts and notes. The content of some of them has come down to us in one way or another but much of it has been lost. About the latter there has been considerable speculation. Many students of Hunter's life believe that some of these unpublished notes might well have anticipated Darwin. It is a fact that there is much in Hunter's published writings that suggests that he had in mind some vague theory of evolution. It is also a fact that Darwin quotes Hunter at considerable length in his own great work.

The manuscripts were for many years in charge of Everard Home. There are different versions as to what use he made of them. In any event, shortly after Hunter's death, Home began a series of brilliant papers which he continued to present over a period of years. Many people said, "He is another Hunter" or "He must have acquired his talent from long association with a great man." Other critics, less kindly disposed, rather more than suggested that they were deliberate plagiarisms from Hunter's unpublished work. It was suggested many times that all of the old manuscripts be assembled and published. Home said it wasn't practical. A little later they were all destroyed by fire. Home said it was an accident. Some folk said it was arson. Nobody knows. Let it rest there.

We are indebted to William Clift, the humble laboratory assistant, for the preservation of some of the unpublished works. During the many years that he worked with Hunter he made copious notes of his own, including many verbatim copies of the master's own notes. Years later these notes were edited and published in two volumes by Richard Owen, a son-in-law of Hunter's, under the title "John Hunter's Essays and Observations on Natural History, Anatomy, Physiology, Psychology and Geology." There is much in this work which is suggestive of the latter work of Darwin and Darwin quoted from it. For this and other things Clift deserves a place in history. Beginning as he did as a humble

laboratory assistant, he became, through his long association with Hunter, a distinguished naturalist himself and his knowledge and ability were often invoked by later writers. In fact, toward the end of his life he was elected to membership in The Royal Society. In later years he bore a striking facial resemblance to his old master. This was frequently commented upon, sometimes in a manner not entirely complimentary to either party. These implications may or may not have been true, at any rate, to illustrate Clift's attitude in the matter, he is said to have remarked upon one occasion when he overheard these aspersions, "If I believed what I know you think, I should not tip my hat to the greatest duke in England."

I approached this discussion with a sense of reverence. I am about to leave it with a sense of utter inadequacy. When one considers that every year and for many years on the fourteenth of February some distinguished surgeon is invited to appear before the Royal College of Surgeons and deliver a eulogy upon Hunter and his life and that each speaker in turn has found some new anecdote to relate, some different accomplishment to praise and some new trait to glorify, it must be obvious that no short effort of mine could do justice to the subject.

I can perhaps no better conclude my few remarks than to quote a short sentence from one of the many letters written to Jenner. Jenner had written asking for suggestions in connection with a problem upon which he was working and speculated at considerable length upon possible explanations. The answer was brief and to the point, "Don't think, try, be patient, be accurate." This was the keynote of Hunter's life. It provided the tempo of scientific investigation for all time.

SURGICAL PROBLEMS

This department, for which the answers have been prepared by competent authorities, is maintained for the benefit of our subscribers. All queries must contain the name and address of the writer. Names will not be published.

Question: What are the indications and the contraindications for the use of the Queckenstedt Test?

Answer: An understanding of the anatomical pathways concerned and the physiological principles involved in the performance of the Queckenstedt Test should leave little doubt regarding its correct clinical application. Partial to complete occlusion of the internal jugular veins in the neck by digital or mechanical compression produces cerebral venous stasis and in turn an increase in the normal intracranial tension. Since the intracranial cavity and the spinal dural envelope, for practical purposes, is a communicating closed space, any change in the intracranial pressure will be simultaneously transmitted to the entire subarachnoid space by a shift of the cerebrospinal fluid. Mensuration of this change may be made by attaching a manometer to a spinal needle introduced into the lumbar thecal sac or into the cisterna magna.

It therefore becomes apparent that in performing the Queckenstedt Test we are concerned with the patency of the internal jugular veins, the great sinuses of the intracranial cavity and the cerebrospinal subarachnoid spaces. The test for the patency of the internal jugular veins or the lateral cerebral sinuses is made in the same manner as the Queckenstedt Test except that the increase in intracranial pressure (as registered by a manometer attached to a needle introduced into the cisterna magna or the lumbar subarachnoid space) following the digital compression of one jugular vein, is compared to the rise of cerebrospinal fluid pressure obtained by compression of the other internal jugular vein. The patency of the cerebral subarachnoid spaces may be determined by measuring the response of cerebrospinal fluid pressure at the level of the cisterna magna following jugular vein compression. This has little application.

The greatest use of the Queckenstedt Test is in determining the patency of the spinal subarachnoid space. Very slight to complete block of this space may be demonstrated if the test is carefully conducted and properly interpreted. It might be said that its only useful clinical application is in disease that affects the contents of the vertebral canal or the region of the foramen magnum. Certainly jugular compression should not be performed on patients with brain trauma, brain abscess or brain tumor. In case of suspected compression lesion of the spinal cord at or above the fifth cervical spinal cord segment, one should exercise great care in performing the test. When the test is carried out in such instances it is inadvisable to withdraw spinal fluid if a block of the aubarachnoid space is demonstrable. The Queckenstedt Test should not be employed routinely as seemingly is being taught to the present day medical students.

Question: Should an eardrum be incised more than once in the presence of an acute otitis media?

Answer: When an eardrum has once been incised or has ruptured spontaneously, and the middle ear has been discharging, further incision is not only valueless, but may be harmful. A diminished or absent discharge and less pain or none at all, indicate that the ear is healing. If the discharge diminishes and the patient suffers ear pain and headache, we must infer that the disease is progressing. Additional incisions will not influence such a condition.

As a rule, the opening which nature has provided by means of a spontaneous perfora-

tion is sufficient for drainage purposes and does not require enlargement by means of incision. Occasionally, when the otitis media is seen in the first few days, such a perforation may be enlarged if the drainage is poor and the opening appears to be too small. Usually even this type of perforation is quite adequate.

A localized bulge of the drum membrane, appearing some days after spontaneous perforation or incision, indicates that an acute mucoperiostitis or osteitis is present. In such a case incision is of no value.

Multiple incisions can cause a permanent perforation or loss of hearing, or both.

Question: Will you kindly recommend a book or monograph on injuries and diseases to the shoulder? I am particularly anxious for a work on traumatic injuries and their aftermath.

Answer: To our mind by far the best book on this subject is *The Shoulder* by E. A. Codman, M.D. of Boston. This book was privately printed and is not sold through agencies or at book-stores. If you desire a copy, write directly to Dr. E. A. Codman, 227 Beacon Street, Boston, Mass., and enclose check for the price of the book, which is ten dollars.

Question: Has anyone written an authorative book on diet and malignancy? If so, what is the title, the author, and when published?

Answer: Cancer and Diet, By Frederick L. Hoffman, LL.D., Published by The Williams & Wilkins Company, Baltimore, 1937.

Question: Within five months a patient of mine will be at term. Her first baby (six years ago) suddenly died without apparent cause on the fifth day of its life. No autopsy was obtained. Fifteen months later she, again, gave birth to an apparent healthy, living child. A month ago it developed a syphilitic keratitis. I took the mother's Wassermann and it was four plus. At this time is it right and proper for me to start anti-syphilitic treatment in the mother?

Answer: Yes, begin treatment at once. Treat the mother, primarily, for the unborn child. Give a treatment each week of neosalvarsan intravenously, and bismuth salicylate intramuscularly.

X-ray the long bones of the baby when about eight days old for pathological changes.

Repeat the mother's Wassermann after delivery, and continue treatment until you are satisfied she is cured.

(We refer you to Dr. William Daily's article on this subject in the February, 1937, number of The American Journal of Surgery. Vol. 35, p. 281. Ed.)

Question: An unmarried virgin woman of 28 years complains of pain across the lower back. By rectal examination one feels an extreme retroversion of the uterus. The tubes and ovaries are apparently normal. Do you think her backache would be improved or cured by operation if the uterus was suspended? Or what do you advise?

Answer: No doubt your patient has a congenital retroversion of the uterus. In the absence of pelvic inflammatory disease this will not cause backache. It is not good practice to operate for congenital retroversion. In your case I'd have an *x*-ray taken and studied. She may have sacro-iliac disease or an arthritic condition of the lower spine.

Question: A male patient complains that every time he has a hard defecation there is an urethral discharge which is gray in color. He has never had any venereal infection. His urine is clear and Wasserman is negative. What can I do to help him?

Answer: The usual cause for urethral discharge following straining at the stool is prostatorrhea. The prostatic secretion is expressed by reason of compression of the prostate by passage of the hard stool. Arrangements should be made to have a smear made of the material. If the contents are definitely prostatic, correct the man's way of living so that he will not be bothered with hard stools. Should the material contain pus in addition to the normal prostatic markings, prostatic massages twice a week are indicated in addition to his dietary corrections.

BOOK REVIEWS

THE MANAGEMENT OF OBSTETRIC DIFFICULTIES. By Paul Titus, M.D. With 314 illustrations, including 4 color plates. St. Louis, The C. V. Mosby Company, 1937. Price $8.50.

We opened this book with keen anticipation because we had met the author, and what is more to the point, we knew his reputation for solid work, and that his was a wide experience in his chosen field. It is not amiss to state that Dr. Titus did not disappoint us. He has done an excellent piece of work. This book will be most useful to the general practitioner who "takes confinement cases," and the obstetric specialist might do well to linger through its pages.

This is not a conventional textbook. Therefore, many chapters that must be included in the formal textbook have purposely been omitted.

The author is of the opinion that abnormal conditions and active operative procedures seem best presented by means of illustrations with concise titles. Where undecided issues arise the author frankly expresses his own views about them and, also, his preference as to procedures. Inasmuch as obstetrics and gynecology interlock so closely, chapters on pelvic floor damage, postpartum uterine misplacements and their treatment, tumor growths complicating pregnancy, ectopic pregnancy, cervical infections, and sterility are given thorough consideration.

The author divides his book into eight sections, the headings of which are: 1. Sterility; 2. Difficulties in Diagnosis of Pregnancy; 3. Complications of Pregnancy; 4. Complications of Labor; 5. Obstetric Operations; 6. Complications of the Puerperium; 7. The Newborn Infant; 8. General, includes Preparation for Obstetric Operations, Prenatal, Postpartum, and Postoperative Care of the Patient, Obstetric Analgesia and Anesthesia, and Intravenous Dextrose Injections, and Blood Transfusion.

This work of 879 pages is well illustrated. Better than that the text is readable and the author has not fallen into the habit of being "stoggy" and giving the impression he is writing for the effect he wishes to create; so common with many authors these days. There is an ample Index.

Anyone who does obstetrics will find it profitable to read this book carefully.

STEDMAN'S PRACTICAL MEDICAL DICTIONARY. By Thomas Lathrop Stedman, A.M., M.D. 1303 large double column pages, 22 plates, 5 in colors, 194 text illustrations, 217 tables, with thumb index. $7.50.

The 1936 thoroughly revised and entirely up-to-date thirteenth edition of this celebrated standard American medical dictionary is now in its 25th year.

It is edited by Dr. Thomas Lathrop Stedman, dean of medical editors and recognized as one of the greatest authorities on medical terminology and lexicology. It is an important reference for everyday use, giving the pronunciation and derivation of more than 15,000 modern medical, surgical, pharmacal, dental, veterinary and other scientific terms, including hundreds of new terms necessary in order to derive full benefit from recent medical literature, medical meetings, and to evade embarrassments in professional conversation.

A special new feature is the chapter on Medical Etymology which aids in the clearer understanding and remembrance of present terms and in coining new terms. The new edition accords with the 1936 U. S. Pharmacopoeia and the National Formulary, with Bergey's bacteriological terminology, with the B.N.A. nomenclature, gives the principal terms of the German N. K. terminology and an appendix supplies the new British Anatomical Nomenclature with B.N.A. equivalents. There is also an index of 217 useful tables. Here is a medical dictionary for constant use on which one can rely and is an ideal reference work for practitioners, students and all connected with the field of medicine in any capacity.

BONES. A STUDY OF THE DEVELOPMENT AND STRUCTURE OF THE VERTEBRAL SKELETON. By P. D. F. Murray, M.A., D.SC.

Cambridge, at the University Press, 1936. Price $2.50.

This small well written book of 203 pages, including the Index, does not attempt to be comprehensive in its survey of the facts and theory of skeletal structure and development, but it clearly discusses certain aspects of the subject, particularly the question of the relation between the structures seen and the morphogenic factors intrinsic in the elements on the one hand, and those, mainly of a mechanical nature, which act upon it from without, on the other. The main mass of the book is in the first four chapters, which deal with the embryonic and postembryonic development and with the structure of the normal and modified bony skeleton considered in relation to the function it fulfills.

The chapter headings are: The Primary Development of the Skeleton; The Development of the Bony Skeleton; Functional Changes in the Forms of Bones; The Mechanical Structure of Cartilage; The Mechanism of Bony Adaption; Summary and Conclusions. Space is devoted to References, and, as said, there is an ample Index. There are forty-four illustrations.

This is a sound and splendid piece of work.

Cosmetic Dermatology. By Herman Goodman, B.S., M.D. New York, McGraw-Hill Book Co., Inc., 1936.

This work, we believe, will prove of interest and be of value to the general practitioner who perforce must do a certain amount of dermatology, and, also, to the man who restricts his work to this field.

S. L. Mayham, in the Foreword writes, "Many months ago Dr. Goodman and I were discussing the effect of radiant energy of the skin. The discussion carried us into the physical, chemical, and physiological properties of a wide variety of cosmetic materials. At the time, I deplored the fact that a good sized library of technical works was needed to insure having all of this information at hand when needed. The next day he brought in for my inspection the most complete dictionary of cosmetic materials it had ever been my good fortune to see. This was the beginning of the present work . . . The text before you now includes the Dictionary of Ingredients and the preliminary work on the physiology and pharmacology of cosmetics . . . "

Naturally, a background of dermatology is necessary to use this work intelligently.

To give an idea what its 54 chapters cover, a few of the chapter headings we selected are: Acne and Facial Blemishes; Antiseptics; Astringents; Bathing; Bleach; Burns; Camphor Ice; Clay; Cleansing Cream; Cold Cream; Corns, Calluses and Warts; Depilatory; Eye Preparations; Face Powder and Rouge: Hair Dyeing; Head and Body Lice; Lead Preparations; Massage Oil; Nail Preparations; Permanent-Wave Preparations; Deodorants; Poison Ivy; Reducing Agents; Scabies; Shaving; Sunburn and Sun Tan; Tissue Cream; Toilet Waters and Odors; Vanishing Cream.

A chapter is devoted to Vitamines and Hormones; Appendix A—Cosmetic Colors; Appendix B—Outline of Perfumery; Appendix C—Weights and Measures. There is an ample bibliography.

A Synopsis of Surgical Anatomy. By Alexander Lee McGregor, M.CH., F.R.C.S. (Eng.). With a foreword by Sir Harold J. Stiles, K.B.E., F.R.C.S. (Edin.). Third edition. Illustrated with 648 figures. Baltimore, William Wood and Company, 1936. Price $6.00.

The medical student and the physician who desire a compact book on surgical anatomy will not go astray in possessing this volume which is now offered in a third edition.

This edition represents a revision of the previous issue, and the text has been amplified by a dozen new figures and a few additional pages of text. The modern surgery of the thyroid gland has necessitated a more detailed consideration of the nerves related to that gland, and the description of the ossification of the clavicle has been harmonized with the work of Fawcett and Mall. The section on hernia of the diaphragm has been amplified considerably. The index has been considerably increased. The "new terminology" has been used throughout the book.

The author in the first edition began his preface, "The book has been written with the object of presenting anatomical facts of practical value to the senior student and practitioner." Throughout the well illustrated pages this promise has been fulfilled.

THE ABRAHAM FLEXNER LECTURES SERIES NUMBER FOUR. LECTURES ON EMBOLISM AND OTHER SURGICAL SUBJECTS. By Gunnar Nystrom, M.D. Published for Vanderbilt University by the Williams & Wilkins Company, Baltimore, 1936. Price $3.

The Abraham Flexner Lectureship was established November 22, 1927, in the School of Medicine of Vanderbilt University through the generosity of Mr. Bernard Flexner of New York City. An adequate endowment made possible these lectures at intervals of two years. The significant objective of the Flexner Lectureship was to contribute to the maintenance of those ideals and standards of scholarship in medical education and research which will bring increasing honor to Vanderbilt University and Abraham Flexner.

The fourth series of these lectures was given by Dr. Gunnar Nystrom, Professor of Surgery, Chief of the Surgical Clinic, and Director of the University Hospital, Uppsala, Sweden. Professor Nystrom is preeminently qualified to deal with each of the subjects discussed in this volume.

The titles of the lectures are: Embolism of the Arteries of the Extremities and Its Treatment; Pulmonary Embolism and Its Surgical Treatment; Swedish Experiences in Combating Appendicitis; The Cytology of Joint Exudates as an Aid to Diagnosis; The Treatment of Medial Fractures of the Collum Femoris.

This small, illustrated volume of 213 pages, may be termed a medical gem. To those who pride themselves on a well balanced medical library, discriminately selected, this work has earned a place. In addition, the contents of the volume are all that any surgeon could desire.

THE PHYSIOLOGY AND PHARMACOLOGY OF THE PITUITARY BODY. By H. B. Van Dyke. The University of Chicago Press, 1936. Price $4.50.

This excellent work by H. B. Van Dyke, Professor of Pharmacology, Peipin Union Medical College, Peipin, China, is one of The University of Chicago Monographs in Medicine under an Editorial Committee composed of Franklin C. McLean, Anton J. Carlson and H. Gideon Wells.

The book is a successful attempt to describe and to evaluate the scientific foundations of our present knowledge of the pituitary body in terms of physiology, pharmacology, and their related sciences.

There is presented an adequately documented account of the experimental work on the pituitary body during the past fifteen years (up to and including 1935). Clinical observations when pertinent are discussed.

The Table of Contents include: The Anatomy of the Pituitary Body; The Effects of Hypophysectomy; The Growth-promoting Hormone of the Pituitary Body; The Gonadotropic Effects of Implants, Extracts, and Secretion of the Pars Glandularis; The Gonadotropic Substances Occurring in Urine, Blood, and Tissues, Particularly During Pregnancy; The Effects of Hormones of the Pituitary Body on the Secretion of Milk; The Interrelationship Between the Pituitary and the Thyroid; The Interrelationship Between the Pars Glandularis and the Adrenals, Pancreas, Parathyroids, and Thymus; Experimental Evidence That the Metabolism of Foodstuffs Depends Partly Upon Internal Secretions of the Pars Glandularis; The Pars Intermedia and the Pars Tuberalis; The Hormonal Regulation of Chromatophores; The Active Principle of the Pars Neuralis; Is There Convincing Evidence That the Pars Neuralis is a Gland of Internal Secretion? The Effects of Extracts of the Pars Neuralis on the Circulatory System and on the Smooth Muscle of Structures Such as the Uterus and Bowel; Other Effects of Extracts; The Effects of Extracts of the Pars Neuralis on the Metabolism of Water, Minerals, Carbohydrates, and Fats; Appendix, Scientific and Commercial Names of Hormones and Hormone Preparations.

This important book of 577 pages devotes 181 pages to Bibliography. There is an ample Index.

PRACTICAL CLINICAL GYNECOLOGY

HENRY C. FALK, M.D., F.A.C.S.

FIFTH INSTALLMENT

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Chapter VIII

STERILITY

What is sterility?

Sterility is the *incapacity* of a male and a female, living a normal sex life, to initiate the reproductive process. A mating which can produce pregnancies even though they do not go to term, is not sterility; this is usually the result of a lowering of the level of fertility. Sterility means the incapacity of a mating to *initiate* the reproductive process.

It has been definitely shown that when a sexually sound cow properly in heat is mated or bred with a genitally sound bull, pregnancy will surely result. This capacity to initiate the reproductive process with a single exposure is known as absolute fertility. The human animal living under the strain and stress of modern times is very rarely so endowed.

However, there is another fact which is frequently overlooked in studying the human female, e.g., the period in the female animal called "heat," which in the human menstrual cycle (Fig. 97) corresponds to the interval period and not to menstruation, as some observers have stated. Since intercourse in the human animal occurs at any time, without reference to the cyclic ovulatory changes in the female, some exposures take place in a relatively infertile period.

That there are various degrees of fertility in the human animal cannot be denied. They can be classified as:

High fertility;
Medium fertility;
Low fertility;
Absolute infertility

Varying degrees of fertility occur in both males and females. A mating between two individuals of low fertility will frequently result in a sterile mating. A mating between an individual in the class of low fertility with one of high fertility will increase the probability of conception.

When a mating occurs between individuals, both of whom are of high rating in the fertility scale and at the proper time in the female ovulatory cycle, there is an approach to the state of absolute fertility seen in normal animals.

The term "*biologic incompatibility*" has been applied to those cases where a mating between two individuals of apparently high fertility, according to all known tests, has not resulted in pregnancy (over an extended period of time, i.e., two years). These individuals sometimes are divorced and in other marriages successful pregnancies result, proving that the first pairing was "biologically incompatible."

As individuals increase in age they become relatively infertile with the result that as they approach the menopause pregnancy does not occur so readily, but should either be exposed to a highly fertile individual pregnancies may occur. Matings are fertile or sterile according to the fertility average established by both.

The gynecologist, in dealing with the sterility problem, must always remember that although the female presents herself as a sterility case it is not a female problem alone but always one of both the male and the female in the mating. If this is disregarded, the number of successes will be very low and the wrong individual will frequently be held responsible for the sterility of the union.

In studying a large series of sterile couples it will be found that there are several factors which may be the cause of the lowering of fertility on the part of either mate; which one of these factors is the paramount or chief cause, may be difficult to prove. In those instances of absolute infertility on the part of either mate, one single factor may be the sole cause. But in cases of relative infertility three or four conditions are usually found, none of which is sufficient to be an absolute cause of infertility, yet in combination they produce a sterile mating.

Some men, in attempting to find the causative factors of the sterile union, place both the man and woman in a hospital, putting them through an exhaustive series of tests. Most

physicians find that the hospitalization of these cases is impractical.

Before beginning the study of any case of sterility it is necessary to explain to the couple that such a study is a lengthy procedure and request their earnest cooperation and patience. No definite opinion as to the etiology in a lowered fertility case should be given until all examinations and tests are completed.

HOW SHALL ONE PROCEED TO DISCOVER THE CAUSES OF A STERILE MATING?

At the first consultation no examination other than a superficial examination of the woman to rule out any gross anatomical lesions which might be the cause of the sterility, such as an intact hymen (no entrance) or absence of vagina, cervix and uterus should be done. If there is no gross lesion which could be the cause for the sterile mating, the husband should be examined before any further examinations are made on the female.

EXAMINATION OF THE MALE

The function of the male in the reproductive act is twofold:

1. He must produce normal spermatozoa in sufficient numbers in the testis.

2. He must deliver these normal sperm in a sufficient amount of prostatic secretion to the vaginal cul-de-sac so they may enter the cervix and uterus (Fig. 98). If the male does this, he is absolved from responsibility.

The various conditions which might prevent the male from fulfilling his responsibility, such as obstruction in the vas, urethral stricture, hypospadias, epispadias, impotence, aspermia are problems for the urologist. As gynecologists our interest is more in the result than in the actual causes. First is the man producing normal sperm in sufficient numbers and secondly, is he delivering them in the proper place for proper function?

Is He Producing Normal Sperm in Sufficient Numbers? What technique shall be used for the collection and examination of the spermatozoa?

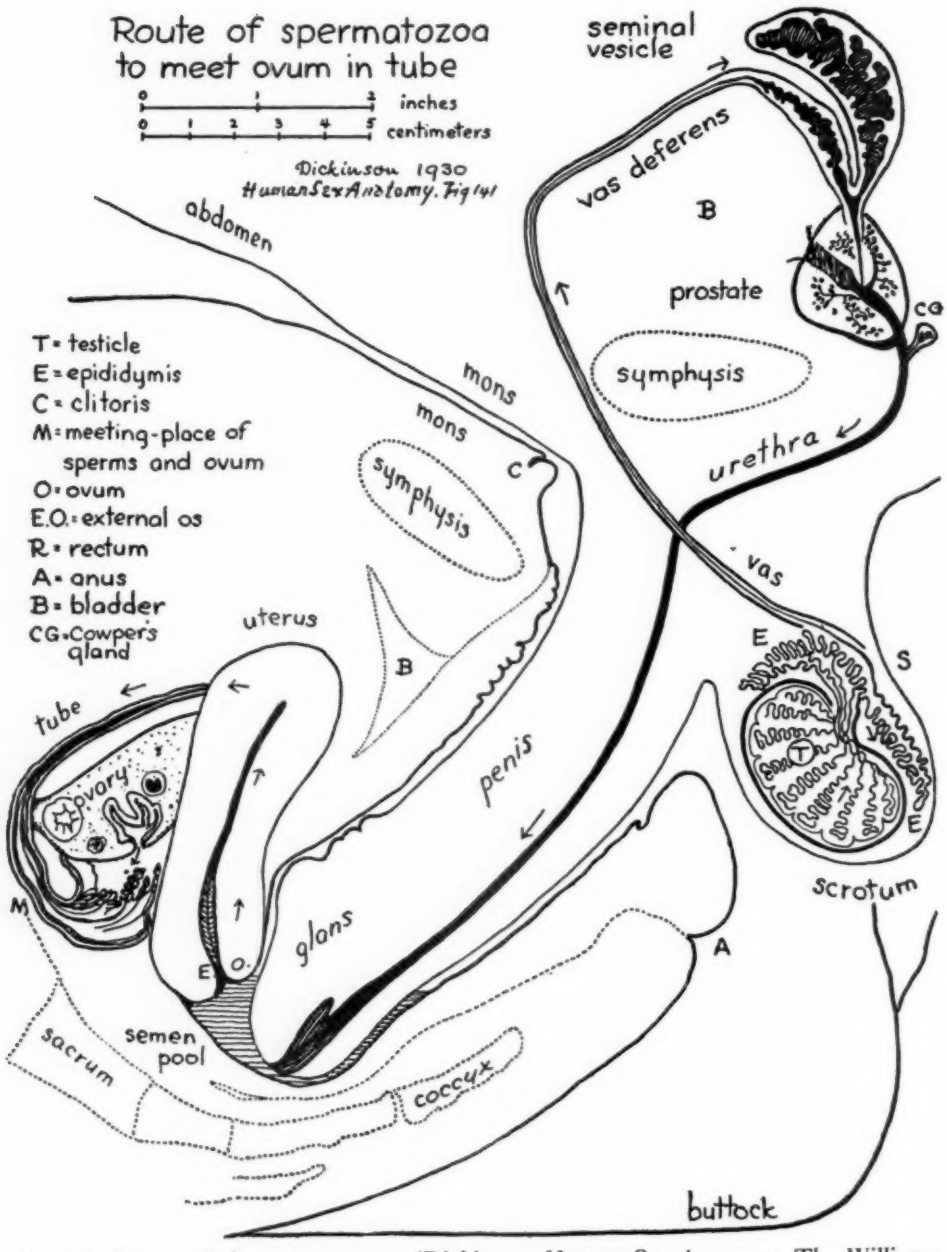

FIG. 98. Route of the spermatozoa. (Dickinson. Human Sex Anatomy. The Williams and Wilkins Co., 1933.)

Three methods may be used:

1. The specimen may be produced by the patient, self-induced, in the office and collected in a clean glass container (a clean wide mouthed bottle or wide test tube can be used).

2. The patient may have intercourse with his wife at home, withdraw and ejaculate the entire amount of his fluid into a wide mouthed bottle and send it to the office or laboratory.

3. A condom specimen may be taken.

The first two methods are preferable. Condom specimens are not as favorable for examination as specimens collected in a clean wide mouthed bottle because (1) some rubbers contain a substance which kills the sperm before any examination can be made even if the powder itself is washed off, and (2), a large amount of foreign material, such as powder, causes improper interpretation of the contents of the seminal fluid. If a condom is used, it must be washed thoroughly so as to free it from all powder. Following intercourse it should be removed immediately and its contents emptied into a glass container and stoppered. Some observers believe this gives the most complete sample.

When the specimen is collected at home the patients should not use absorbent cotton as a stopper for the bottle, instead they should use the regulation metal screw cap or an ordinary cork. The specimen should be brought to the office or laboratory in the handbag. The use of external heat in the transportation of the specimen is unnecessary and probably harmful. One of the reasons given for the testis being in the scrotum is that spermatozoa do not live long at body temperature as the heat of the body itself is sometimes enough to destroy them. This is also the explanation for sterility in cases of undescended testis. Male rabbits can be made sterile by having the scrotum kept in hot water (104 degrees F.) for a period of time.

Gross Examination of the Spermatozoa. The specimen should be examined within two hours after emission. The *amount* of ejaculate is usually about 3.5 c.c. The older the individual, the smaller the amount; during sexual excesses the quantity of the ejaculate is markedly diminished. It is not

unusual to find in young married couples having excessive sexual relations, 0.5 c.c. of fluid in the ejaculate. The quantity of the ejaculate is very important as the major part of the fluid content is from the prostate and seminal vesicles. It has been shown by Kurzrock that the prostatic fluid is the substance which makes the cervical plug more permeable to the spermatozoa.

Color. It is *opaque* and *grayish* in color. At first it is quite *viscid* and *tough*, but liquefies after standing for three-quarters of an hour. A similar change takes place in the vagina. Patients examined immediately after intercourse show a thick, tenacious material in the cul-de-sac. In one-half hour this material becomes quite fluid. This was thought to be due to the acid secretion in the vagina, but as it occurs in the bottle also, it must be inherent in the ejaculate itself.

Microscopic Examination. The cell count. Method of counting the spermatozoa. A white blood counting pipette is taken and a measured quantity of the ejaculate is diluted* 1–100 in the pipette. This is thoroughly mixed and the number of spermatozoa in one square millimeter in the counting chamber is computed. This number is multiplied by 100 for the dilution, by 10 for the depth of the chamber and by 1000 to convert it into cubic centimeters.

At first an actual count of the spermatozoa is necessary. One will find, however, as one gains experience that the actual cell count is not so essential. One learns to recognize when examining a slide with motile sperm whether or not they are in the millions. In a normal specimen there should be at least 70,000,000 spermatozoa per c.c. and a normal ejaculate should contain approximately 200 million spermatozoa. When they are deficient in number there are usually other abnormalities present, and by evaluating these other abnormalities, one can judge the fertility of a particular specimen.

* Diluent.

Sodium bicarbonate	5.0
Formalin	1.0
Water q.s. ad	100.0

Duration of Motility. A thick smear is made and a layer of vaseline is placed around the edges of the cover slip to prevent drying. The slide is kept at normal room temperature. An examination of a specimen from a man of high fertility will show large numbers of spermatozoa hurrying about the field in an apparently purposeful activity. They will usually move across the entire field in about eight seconds. It has been established that spermatozoa travel from 1 to 2 cm. in about eight minutes. Sperm activity should continue on the slide of a normal specimen for about two to three hours. Some observers prefer to study sperm motility by taking a fresh drop from the bottle every hour for five hours. A normal specimen examined this way should show motility for at least this length of time.

In the past this generally constituted the entire examination. The doctor felt that if he saw motile spermatozoa on a slide it absolved the male from all responsibility in the sterility. But this is not enough; the man must also produce enough prostatic secretion, must deliver the sperm in the proper place and normal spermatozoa must be present in sufficient numbers.

Morphology. To study the morphology of the sperm a thin smear of the specimen is made, fixed with Schaudinn's solution and stained. The technique described is that used by Cary and Hotchkiss.

1. Dry the slide in air.
2. Immerse it for one minute in 7 per cent corrosive mercuric chloride (2 parts) and absolute alcohol (1 part).
3. Immerse for one-half minute in 50 per cent alcohol.
4. Immerse for one-half minute in distilled water (3 ounces), tincture of Iodine (2 drops).
5. Wash in tap water.
6. Immerse for one-half minute in aqueous solution of 5 per cent eosin.
7. Immerse for one minute in 50 per cent alcohol (3 ounces) and concentrated hydrochloric acid (2 drops).
8. Wash in distilled water.
9. Immerse for two and one-half minutes in hematoxylin.
10. Immerse for one minute in distilled water (3 ounces) and glacial acetic acid (2 drops).

11. Wash in distilled water.
12. Dry and mount.

This method stains the nuclei blue and the cytoplasm red.

For quick examinations a thin film may be fixed on the slide with Schaudinn's solution or in the flame and stained with Loeffler's methyline blue or aqueous gentian violet one-half per cent.

Moench who has made an exhaustive study of the morphology of the human sperm has described more than fifty forms.

A normal spermatozoon has a round head (5 by 3 microns) with fairly sharp shoulders, a short body and a longer tail (about 55 microns long). Many variations or abnormal forms are found such as (Fig. 99):

1. Tapering head;
2. Narrow head;
3. Apparently normal heads without tails;
4. Normal heads with a very short tail;
5. Double heads.

For a while the double heads were considered artefacts but by microdissection Moench has definitely shown that they are not artefacts (not one spermatozoon superimposed upon another), but are actually double headed spermatozoa.

6. Multinucleated heads;
7. Double tails, etc.

Differential cell counts should be made on stained specimens and from 300 to 500 spermatozoa should be counted. The number of normal and abnormal forms should be thoroughly checked. This is very important because if 25 per cent of the cells counted consist of abnormal forms, the male must generally be classified as infertile.

Even though the individual discharges 100,000,000 spermatozoa of which 25,000,000 are abnormal forms and 75,000,000 are normal forms, he must be considered in the infertility group. One should never specifically state to the patient or his wife that he is sterile, because if subsequently his sperm should improve and impregnantion result, explanations are difficult.

As Kleegman states,

It is a great advantage for the gynecologist to learn this more accurate and more complete appraisal of the semen . . . knowledge of sperm mor-

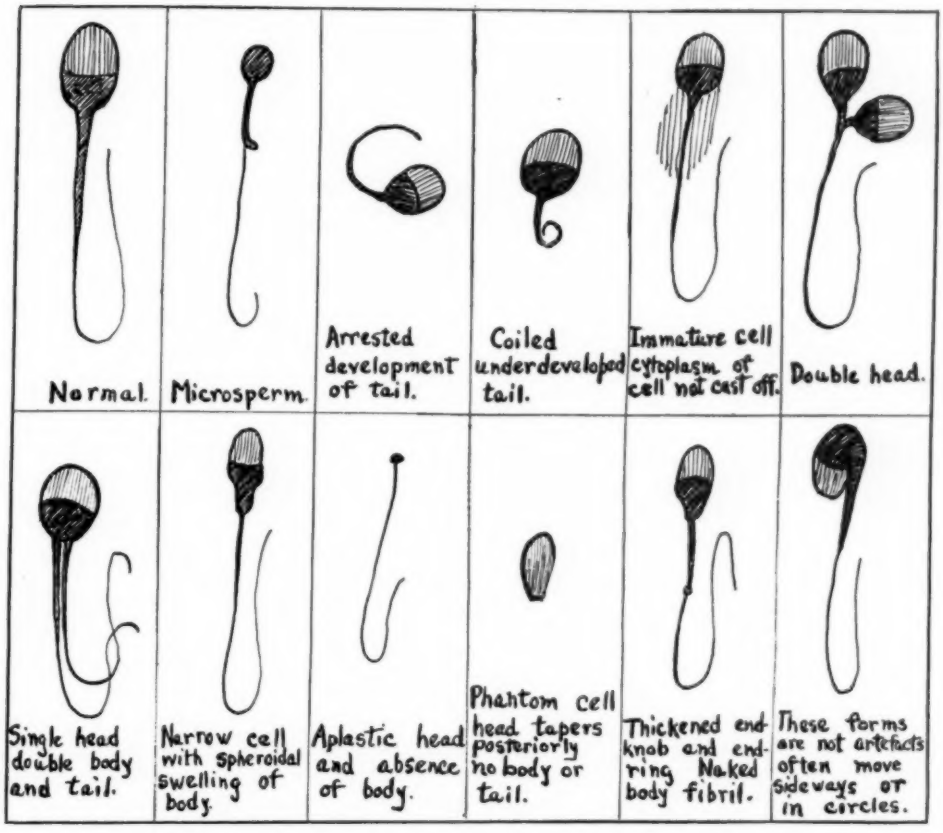
Normal.
Microsperm.
Arrested development of tail.
Coiled underdeveloped tail.
Immature cell cytoplasm of cell not cast off.
Double head.
Single head double body and tail.
Narrow cell with spheroidal swelling of body.
Aplastic head and absence of body.
Phantom cell head tapers posteriorly no body or tail.
Thickened end knob and end-ring Naked body fibril.
These forms are not artefacts often move sideways or in circles.

FIG. 99. Microscopic appearance of normal and some abnormal spermatozoa.

phology cannot be acquired in one session by seeing a few pictures or slides but requires considerable time and patience. It is preferable to start by first studying a number of known fertile sperm specimens using Moench's writings as guide to such study, thereby learning first what the normal is, before undertaking to assay the value of an unknown specimen.

Abnormalities in the Spermatozoa. The abnormalities in the spermatozoa that may be found are:

1. Lowered motility. Some of the sperms are not as motile as they should be. The cells do not move across the field rapidly enough.

2. Duration of motility is diminished.

3. Their number is diminished.

4. Abnormal morphology. There is an increase in the number of abnormal sperm.

5. Round cells, leucocytes, cell debris and crystals when found indicate an abnormal prostatic secretion, usually the late result of an infection.

If any of these are found, one is probably dealing with an abnormal specimen.

HOW CAN A LOW FERTILITY OR HIGHLY ABNORMAL SEMEN BE REMEDIED?

Very little is known about this problem, as yet most of the treatment is empirical. When an abnormal semen specimen is found the male should have a complete endocrinologic work-up to ascertain whether he may be suffering from an endocrine imbalance. Thyroid substance, 2.5 to 5 grains, or thyroxin, grain $\frac{1}{320}$ to $\frac{1}{160}$, may be used depending upon the extent of the lowering of the basal metabolic rate. If he is overweight he should be put on a physical culture regime: fresh air, regular living and exercise. Sex rest should be used, particularly in those cases where the spermatozoa are normal in appearance but reduced in numbers. Intercourse should be allowed only during that period when ovulation is most likely to occur, e.g. the twelfth to nineteenth day after the first day of menstruation. The female will thus be exposed to the male during the period of her greatest fecundity. Prostatic massage, heat to the prostate, etc., are empirical but seem to have some beneficial effect. It is particularly indicated in those cases where the physical examination shows an enlargement of the prostate and a smear shows pus cells in the prostatic secretion. The value of testicular extracts are as yet unproved; theoretically they are not indicated; experimentally they have been found valuable in preserving secondary sex characteristics following castration. When used in animals with testes they seem to have a definite toxic effect on the testicular epithelium. The prolan

B-like hormone obtained from pregnancy urine, 1 c.c. triweekly, in some cases seems to have a stimulating effect on the testes and seems to increase the number of normal spermatozoa.

If the physical examination of the male discloses organic lesions, such as undescended testes, varicocele, etc. these may be corrected surgically. In cases showing an aspermia, obstructive lesions of the vas, urethra or epididymis may be found. All of these phases of the problem belong primarily to the urologist and not to the gynecologist. Both should work together on the sterility problem when the male is at fault.

Is He Delivering the Spermatozoa in the Proper Place for the Insemination of the Cervix? Even though it has been definitely established that the spermatozoa are normal, the male has not yet completely fulfilled his part of the contract in the mating. He must be able to deposit these spermatozoa in the seminal pool of the cul-de-sac of the vagina.

In What Conditions Does This Not Occur? In hypospadias, premature ejaculation, non-penetration, etc. In order to prove this, a Huehner test is performed.

Technique of the Huehner Test: This test consists in the examination of the fluid in the seminal pool in the vagina and of the mucus plug in the cervical canal following intercourse.

A. Examination of the seminal pool.

Within an hour following intercourse the patient should present herself at the doctor's office. A speculum is introduced into the vagina without lubricant, as a lubricant may destroy the motility of the spermatozoa, and a drop of fluid from the seminal pool obtained with a syringe (Fig. 100) is placed on a slide, covered with a cover slip and studied. The slide in a normal case will show: (Fig. 101).

1. A large number of squamous epithelial cells;
2. Some leucocytes;
3. Cell debris;
4. Active spermatozoa.

The number and condition of the spermatozoa will depend upon:

1. Male production and his ability to place the ejaculate in the seminal pool.
2. Hostility of the vagina.

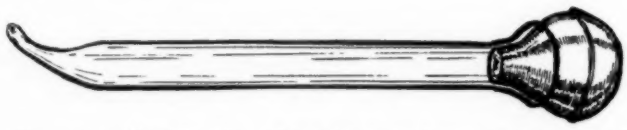

FIG. 100. Syringe for obtaining specimen from seminal pool and cervix.

The vagina is normally acid and the spermatozoa live in a slightly alkaline medium. Following ejaculation into the cul-de-sac the spermatozoa seem to rush to the cervical canal as quickly as possible. Cases have been observed where an acid vaginal secretion has destroyed all motility in the

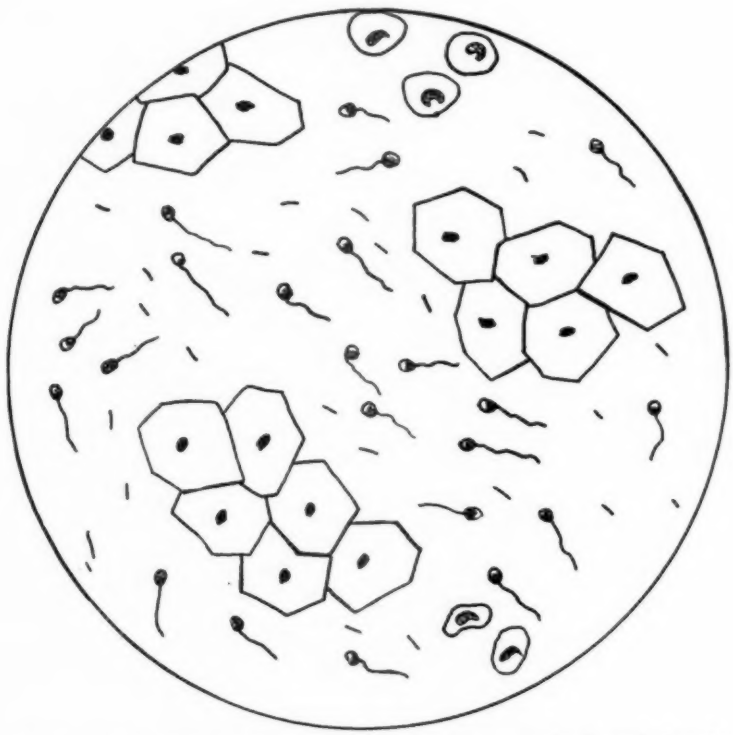

FIG. 101. Microscopic appearance of fluid from seminal pool shortly after intercourse.

spermatozoa of a normal specimen fifteen minutes after intercourse and yet, some observers regard the reaction of the vagina as unimportant.

3. Length of time after intercourse that examination is made.

In a normal mating no live spermatozoa may be found in the seminal pool three to five hours after intercourse.

4. Volume of the ejaculate.

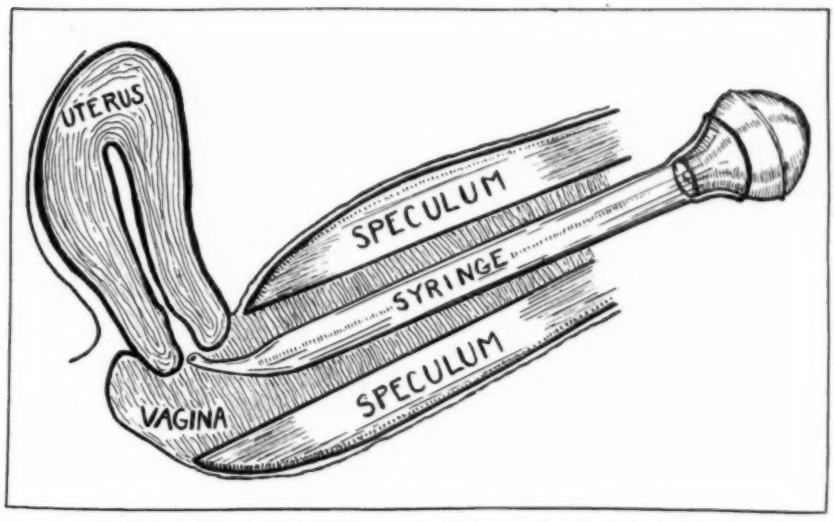

FIG. 102. Method of obtaining specimen from cervical canal.

This may be very important. Sexual excesses cause a reduction in the quantity of prostatic fluid secreted. As a result, specimens are seen with normal spermatozoa and a reduced amount of prostatic fluid. This small amount of alkaline prostatic fluid will not be sufficient to protect the spermatozoa from the acid vaginal secretion and their motility is destroyed very early.

5. The portion of the seminal pool which is examined.

Different portions of the pool will show a different spermatozoa content.

B. Examination of the cervical contents.

Technique. After the seminal pool fluid has been examined, the cul-de-sac is wiped absolutely dry. The outer portion of the cervix is also completely dried. With a pipette, (Fig. 102) loop or spoon curette, some of the thick cervical secretion is obtained, placed on a slide, covered with a cover slip and examined immediately. Great care should be exercised so as not to contaminate the specimen with blood. In the normal case this slide should show:

1. Spermatozoa, are fewer than on the slide of the seminal pool;
2. Some mucus;
3. Occasional epithelial cell or leucocyte;
4. Cell debris.

The presence of living and apparently normal spermatozoa in the cervical canal, in a mating in which the bottle examination of the male ejaculate was within normal limits, absolves the male of responsibility for the sterility.

EXAMINATION OF THE FEMALE

In the male an examination of the semen gives direct evidence as to his probable fertility. In the female there is no single parallel test. Therefore one must draw conclusions and inferences based upon (a) careful history, (b) careful examination and (c) laboratory data.*

History. It is advisable in obtaining the history to question the husband and wife separately.

Marriage History. The marriage history is very important. Women may present themselves three months after marriage and want to know why they are not pregnant. Previous marriages, pregnancies or miscarriages in or out of wedlock should be known. Patients will frequently describe what they considered early miscarriages. They are so anxious to become pregnant that if they miss a period, they immediately ascribe it to a pregnancy. *A missed period is not prima facie evidence of pregnancy.* Missed periods, even in a normally menstruating woman, are caused by many other conditions besides pregnancy.

The Sex Life. Frequency and spacing of intercourse, libido, postcoital satisfaction, etc. Too frequent intercourse may cause a lowering in the vitality of the male ejaculate. The history of libido, etc., must be obtained from the woman; the female view of her sex life is frequently quite different from the male impression. Female libido, however, is not a true index of ovarian function. Patients in whom complete sex life has been established, who as a result of some pathological condition have had both ovaries, tubes and the uterus removed, may continue to have normal libido.

* In this we follow closely the excellent routine laid down by S. R. Meaker in Human Sterility. Williams & Wilkins Co. Baltimore, 1934.

History of Any Abdominal Inflammation. A history of any form of pelvic infection is important; an old appendicitis or inflammation of the bowel may mean previous pelvic inflammatory disease. It is necessary to go over the history very carefully with the woman alone, so that she will give the facts as they are and not as she wishes her husband to believe them.

History of Operations. All previous operations must be carefully reviewed. It is ridiculous to make a study of a sterile mating when the female has had a bilateral salpingectomy.

Menstrual History. When and how it began and what was its behavior? How was it during adolescence? Irregular? What subsequent change of habit developed? Did she suddenly cease having normal periods? What is the present menstrual habit? It is essential to find out from the woman everything that one can with regard to her menstruation in order to establish, if possible, whether or not a normal ovarian function exists. When the menstrual history shows marked irregularity, one may assume the possibility of some developmental arrest in the female.

In the absence of a previous history of a definite pathological process, functional menstrual disorders are usually associated with some form of ovarian deficiency. The management of these conditions is discussed in Chapter VII.

History of Leucorrhea. Leucorrhea in itself may mean very little. Some patients with very profuse leucorrhea become pregnant quite regularly. It is of interest in that it may be the clinical manifestation of an existing endocervicitis, which may interfere with normal insemination. Several patients with sterility of long standing were observed in the endocervicitis clinic who promptly become pregnant after the cervical lesion was cured. This may have been due to a change in the nature of the cervical plug, making it more permeable to the spermatozoa or to a reduction in the leucorrhea.

History of Changes in Weight. It is very important to go into the weight history of the patient. Today when many women try so hard to become slender, irrespective of their normal physiological tendencies, irreparable endocrinologic

imbalance may result. Some of the most difficult sterility problems at the present time are young women, now amenorrheic, who during their adolescence thought they were overweight and went on very strict diets. As a result there was an improper distribution of carbohydrates and proteins. This in turn caused some change in the development of the organs of internal secretion with a resulting imbalance. The exact diagnosis of this imbalance and its correction is frequently very difficult.

Patients may come as sterility studies stating that since their marriage, they have gained thirty pounds. Such a gain in weight must be carefully studied, to establish if possible, some correlation between the gain in weight and a possible endocrine disturbance (see p. 189).

GYNECOLOGICAL EXAMINATION

1. Look for abnormalities, deformities or malformations such as strictures of the vagina found usually in the vault, double vagina, etc. Any conditions which might prevent or interfere with normal intercourse, such as a rigid hymen which makes penetration impossible, in spite of a history of a satisfactory sex life.

2. *Infantilism or Juvenilism* (the latter is the better term). A great many patients may have stigmas of juvenilism and yet not be infantile. They may have long narrow vaginas, underdeveloped uterii, and yet give a normal menstrual history.

Juvenilism can be recognized, according to Frank, by the following factors:

a. High symphysis;
b. Narrow pubic arch;
c. Slender pubic rami;
d. Undue prominence of the vestibular area with a thick rigid perineum;
e. Short underdeveloped vagina with shallow fornices;
f. The cervix projects into the vagina only a very short distance;
g. There is a long, narrow supravaginal cervical segment;
h. The uterosacral ligaments are short and inelastic.

Some men use the "uterine index" to establish juvenilism. They measure the length of the cervical canal and compare it with the length of the uterus. In cases of juvenilism the cervical canal is longer than the uterus itself.

3. *The Cervix.* Normally the cervix is in contact with the posterior vaginal wall and lodges in the vaginal cul-de-sac, or seminal pool (Fig. 103A). Should the cervix be directed forward (fundus retroflexed or retroverted), it would not lie in the seminal pool following intercourse. When the cervix does not rest in the seminal pool it may be a factor in the sterility. The Huehner test should show this. Intercourse in different positions so as to bring the ejaculate in contact with the cervix may be necessary.

In cases of juvenile stigmas, the cervix may be almost flush with the vault of the vagina and there may be no cul-de-sac (Fig. 103B); these patients complain that the ejaculate flows out immediately after intercourse.

The secretion of the cervical canal should be tested with litmus paper to see that it is slightly alkaline. A cervical smear is also taken, as thick mucopus is not a good medium for the spermatozoa to pass through. A thick cervical plug may be a factor in causing a lowering of fertility by preventing the ascent of the spermatozoa. Chronic endocervicitis is one of the most common factors in lowering fertility. Simply removing the plug and treating the endocervicitis has cured many cases of long standing sterility.

4. *Palpable or Visible Pelvic Pathology.* Any palpable or visible pelvic pathology may have an influence or bearing upon the sterility problem and must be considered as an etiological factor in its proper place in the sterility scale.

5. *Examination of the Uterus.* Excepting infantilism, retroflexion or palpable uterine pathology, such as fibroids, the uterus is practically absolved from any responsibility as a factor in the lowering of fertility.

6. *Examination of the Tubes.* The most frequent cause of sterile mating in a woman who is ovulating is some pathological change in her tubes. The recognition and diagnosis

of this pathology is not always easy because the absence of a palpable lesion does not always rule out tubal pathology. Previous to the use of tubal insufflation as a test for patency of

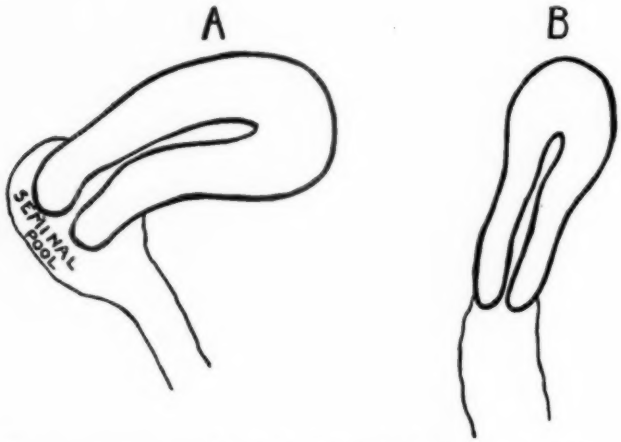

FIG. 103. A, normal cervix in seminal pool; B, underdeveloped cervix and vagina, no seminal pool present.

the tubes, a laparotomy had to be performed to determine the condition of the tubes. Since the development of the tubal insufflation by I. C. Rubin, this is not necessary.

Tubal insufflation, when properly used with strict observance of the contraindications is practically without danger. It should not be done at any time during the menstrual cycle, but should be done seven to ten days after menstruation has ceased (Rubin). At that time the endometrium is again lined with epithelium. The patient should not have had intercourse since the end of her last period, so that there will be no possibility of an early pregnancy.

Tubal insufflation is contraindicated in:

a. The presence of acute, subacute or subsiding inflammation;

b. In the presence of tubal masses;

c. In any uterine bleeding be it functional, menstrual, pathological or after curettage;

d. In chronic endocervicitis with profuse discharge. The latter should first be cleared up. Simple chronic endocervicitis with mild discharge which can be thoroughly cleaned out and sterilized with an antiseptic need not be considered a contraindication.

e. In patients who are suffering from any condition which would make them poor risks for pregnancy, as cardiac disease, pulmonary disease, etc.

TUBAL INSUFFLATION

Patency of the tube can be demonstrated with a Davidson bulb syringe and an intrauterine cannula. In order to obtain the best results it is advisable to use an apparatus especially adapted for the procedure (Fig. 104). This apparatus will deliver carbon dioxide* in a constant steady slow stream to the patient. Some men (Rubin, Meaker) in order to make more precise readings and refinements in diagnosis prefer to have a kymograph register a tracing of the pressure used in the insufflation.

Technique. The cervix is well exposed with a sterile bivalved speculum under a good light. The cervix and its canal are thoroughly cleaned and painted with iodine. The direction of the uterine cavity must be known so that the cannula can be introduced beyond the internal os. *Be very careful not to puncture a retroflexed uterus.* The cervix is grasped with a tenaculum and the cannula, F, is inserted, the rubber plug fitting snugly into the cervical canal. The reducing valve, B, is closed until the carbon dioxide tank, A, valve, D, is opened widely, thus insuring a steady flow of gas. The compression clamp, H, is closed and the reducing valve, B, is then slowly turned until the gauge, K, reads 10 pounds. At this point the compression clamp, H, is slowly released until the gas bubbles slowly through the "bubbleometer," C, at about three pulsations a minute. One may then pinch the rubber tubing, G, and watch the mercury in the manometer, E, ascend. This should mount very slowly, it should require fifteen seconds for the mercury to be raised 100 mm. If the flow is too rapid, a spasm of the tubes may result with improper readings. Tubing, G, from the apparatus is then attached to the end of the cannula in the uterus and the gas is allowed to flow into the uterine cavity. Manometric readings are immediately begun.

*Carbon dioxide is used in preference to oxygen because it is absorbed more rapidly.

If the tubes are not patent the mercury in the manometer will gradually rise. When it registers 150 to 170 mm. of mercury the release valve, J, should be opened or the cannula with-

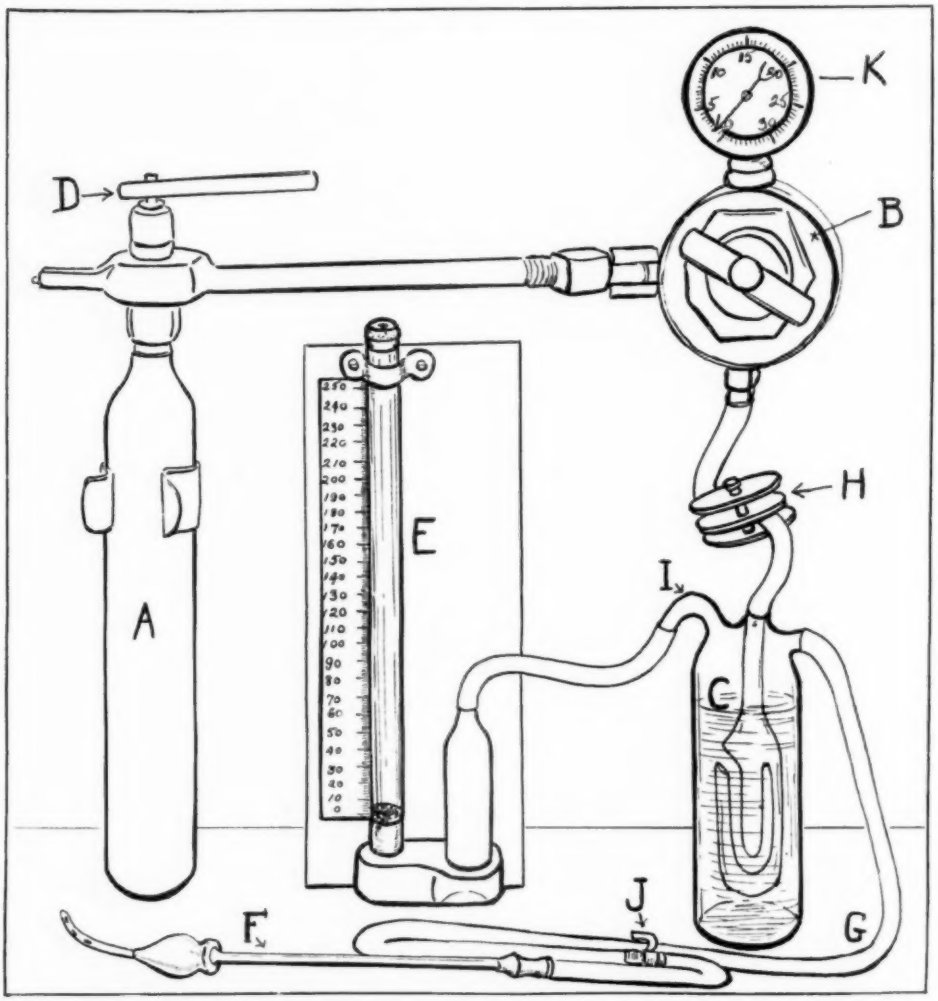

FIG. 104. Tubal insufflation apparatus. A, carbon dioxide pressure tank (1000 lbs.); B, reducing valve to obtain 10 lbs. pressure; C, 40 cc. "bubbleometer" to measure the volume of gas allowed to enter through the tubes; D, valve on pressure tank; E, mercury manometer; F, intra-uterine cannula with olive shaped rubber plug; G, pressure tubing; H, compression clamp; I, outlet to manometer; J, release valve; K, pressure gauge.

drawn. On re-inserting the cannula a second time the pressure may be allowed to rise slowly to 200 mm. of mercury. Beyond that, one cannot go safely, as closed tubes may be ruptured. If the tubes are patent the manometer reading gradually

increases as the carbon dioxide is allowed to flow into the uterus until it reaches 80 to 120 mm. of mercury, when the mercury will suddenly drop 20 to 30 mm. and then gradually rise again to the 100 mm. mark. Sixty to 100 c.c. of carbon dioxide is allowed to flow through the tubes; each bubble in the "bubbleometer" measures about 40 c.c.

At the conclusion of the test the cannula is withdrawn and the patient is allowed to sit up. If the gas has escaped through the tubes into the peritoneal cavity the gas will ascend under the diaphragm and the patient will complain of pain in the shoulders, which may be very severe. In such cases the patient is placed in the Trendelenburg position, following which the pain promptly ceases.

If the first test shows the tubes to be closed, it may be repeated in two to three days. Some men do not like to repeat the test in the same month. They have the patient return seven days after the next menstrual period, using the same precautions as on the first test.

In the second test the patient should be given atropine to overcome any spasm of the tubes which may have been the cause of a false reading. One-half hour before the insufflation $\frac{1}{150}$ of a grain of atropine is given hypodermatically or two hours before the test a $\frac{1}{100}$ grain tablet of atropine is given the patient to place under her tongue and a $\frac{1}{200}$ grain tablet is given by mouth one hour before the test.

If repeated insufflations show the tubes to be closed and the patient is anxious to have their patency restored, it is necessary for surgical therapeusis to know the exact location of the obstruction. For this purpose an opaque (lipiodol) substance is injected into the uterus and the tubes, and *x*-ray photographs are taken for study. For this purpose the complicated set-up of tubal insufflation is not necessary.

Instruments.

1. Good light
2. *X*-ray apparatus with a Bucky diaphragm.
3. Speculum

4. Tenaculum
5. Iodine
6. Sterile gloves
7. Uterine cannula with olive rubber tip and Luer-lock attachment to fit syringe.
8. 10 c.c. Luer-lock syringe
9. Lipiodol (warmed).

The injection is made at the same time in the menstrual cycle as tubal insufflation and the same contraindications and precautions observed as in tubal insufflation, otherwise pelvic peritonitis or abscess formation may result.

The patient is placed on the *x*-ray table with her buttocks well down (lithotomy position) over the *x*-ray plate and the Bucky diaphragm. A sterile speculum is inserted into the vagina and the cervix is thoroughly cleaned and dried. The cervical canal and the cervix is painted with iodine. It is then grasped with a tenaculum forceps and the uterine cannula with the olive rubber tip is inserted into the cavity of the uterus. Warmed lipiodol is sucked into the syringe; the syringe is attached to the cannula and holding the olive rubber tip in close contact with the cervix, the solution is slowly injected into the uterine cavity. The pressure used is gauged by the reaction of the patient, 5 to 6 c.c. are usually enough to distend the uterine cavity and overflow into the tubes. After 4 to 5 c.c. are introduced the patient usually complains of pain either on the right or the left side. If this occurs, the first *x*-ray picture is taken and immediately developed to see whether or not the tubes have been entered and whether the uterus has been completely filled. If the patient is not uncomfortable or if the *x*-ray shows the lipiodol has not entered the tubes more lipiodol may be injected to a total of 6 to 10 c.c. and more pictures are taken. Following this the patient is instructed to return in twenty-four hours when another picture is taken.

Interpretation. If the uterus is completely filled with lipiodol and neither tube is visualized, (Fig. 105) then the tubes are closed at the uterine end. If fluid enters the tubes, it shows itself as a wavy line extending from the cornu of the uterus

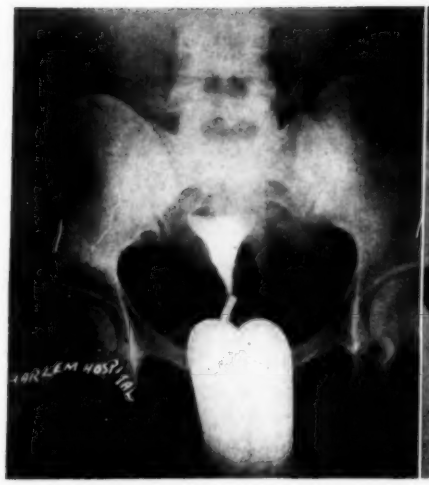

FIG. 105. Uterine end of both tubes closed to lipiodol.

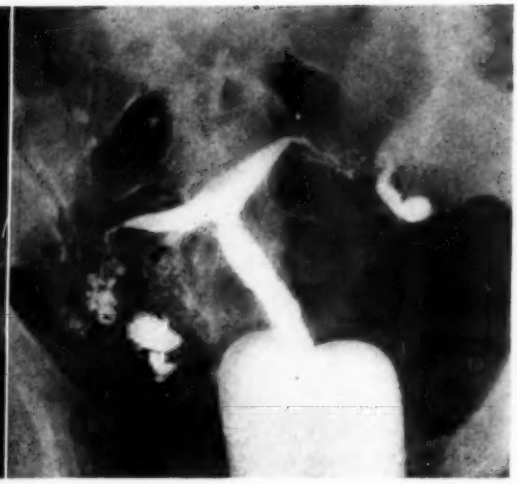

FIG. 106. Right side tube patent lipiodol droplets in the peritoneum. Left side fimbriated end of tube closed-end of the tube bulbous.

FIG. 107. Twenty-four hour picture. Lipiodol still in bulbous end of tube.

FIG. 108. Twenty-four hour picture. Lipiodol free in peritoneal cavity.

outwards (Fig. 106). If the tube is closed at its fimbriated end, the end of the tube usually shows itself to be bulbous (Fig. 106). If the fimbriated end of the tube is patent the lipiodol can be seen in droplets falling into the peritoneal cavity (Fig. 106). In the twenty-four hour picture if the tube is closed some lipiodol can still be seen in the bulbous tip of the tube (Fig. 107). If the lipiodol is seen to be free in the peritoneal cavity, one or both tubes are patent (Fig. 108).

SURGERY OF STERILITY

It is very important before considering any surgical procedure in female sterility, to realize that the permanent restoration of the patency of an occluded tube by surgical measures is successful only in a limited number of cases. It is not at all unusual to operate, restore the patency of the tubes and in examining the patient some months later, find the tubes closed again. It must also be understood that even though permanent patency of the tube is established, pregnancy may not occur. One cannot guarantee the patient that pregnancy will ultimately follow any surgical procedure. All one can attempt to do with surgery, and even this cannot always be promised, is to make impregnation a possibility. The results of surgery are not too promising.

Surgery should not be undertaken until certain factors have been definitely established:

1. That the husband can fulfil all the requirements previously outlined.
2. That the woman is in the childbearing age and possesses her uterus and ovaries.
3. That both tubes are closed to both air and lipiodol.
4. That the pelvic infection which was the etiological factor in the sterility is entirely cured.
5. That all cervical infection is cured.

When Are These Patients to Be Operated Upon? One of the most frequent errors made in the surgical management of sterility is to disregard the cervix. Should the patency of the tubes be restored in the presence of an existing cervical infection, it is still possible that the patient may develop another

salpingitis with all the previous sequels. All patients on whom surgery for restoration of patency of the tubes is to be carried out should have all cervical infection cleared up *before, not after,* the surgical procedures are done.

No surgery should be attempted on any case of sterility until one is sure that all infection both in the tubes and the cervix has disappeared. If there is any question of infection in the pelvis, no form of surgery should be attempted.

INSTRUMENTS

Besides the usual dissection set *special instruments* are required:

1. Fine intestinal needles, small full curved.
2. Very fine mosquito forceps hemostats.
3. Fine suture material, chromic catgut No. 0 or No. 00.
4. Ureteral catheter.
5. Sovak reamer (Fig. 113A).
6. Bonney clamp (Fig. 109).
7. Special types of bulb syringe, with a straight tip and one with a right angle bend in its tip. The tips of all syringes should be bulbous (Fig. 110A).
8. Fine manicure or eye scissors, straight and curved.

WHAT TYPE OF SURGICAL PROCEDURE IS TO BE PERFORMED?

This cannot be decided upon even after lipiodol *x*-ray examination until the abdomen is opened and the exact pathology and point of obstruction in the tube identified.

There are a limited number of procedures that can be performed. The indications and technique for each of these procedures will be given.

PROCEDURE 1. RESECTION, RE-IMPLANTATION OF TUBE INTO UTERUS FOR ATRESIA OF UTERINE END OF THE TUBE.

X-ray examination with lipiodol before admission to the hospital shows a normal uterine shadow with both horns of the uterus closed.

Step 1. The abdomen is opened with the usual left paramedian incision. The intestines are thoroughly walled off, three laparotomy pad technique. The pelvic structures are examined and the factors identified and separated.

All adhesions are cut using sharp dissection, not broken with fingers, careful hemostasis being observed. If sharp dissection and careful hemostasis are not used, a small amount of

FIG. 109. Bonney Clamp. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

bleeding will occur, which hemorrhage may cause new adhesions to form. These adhesions may cause a closure of the fimbriated end of the tube by postoperative adhesions.

Step 2. Preoperatively it was shown that an obstruction was present at the uterine horn. It is now necessary to prove the extent of the patency in the distal end of the tube. To do this deliver the fimbriated end of the tube into the wound and with a syringe force air through the tube (Fig. 110 B, C). The bulbous tip of either the straight or right angle syringe is introduced into the fimbriated end of the tube and held there by an Allis clamp or the fingers. Air is then forced into the tube. The tube distends and various bulbous areas appear, being most prominent behind the point of obstruction, and beyond that the tube is straight and narrow. The obstruction is therefore between the distended portion of the tube and the uterine cornu.

Step 3. The obstructed uterine end of the tube is excised, with care to keep close to the tube so as not to interfere with the vessels of the broad ligament. (Figs. 111, 112.) The excision of the tube is begun at a point just proximal to the point of

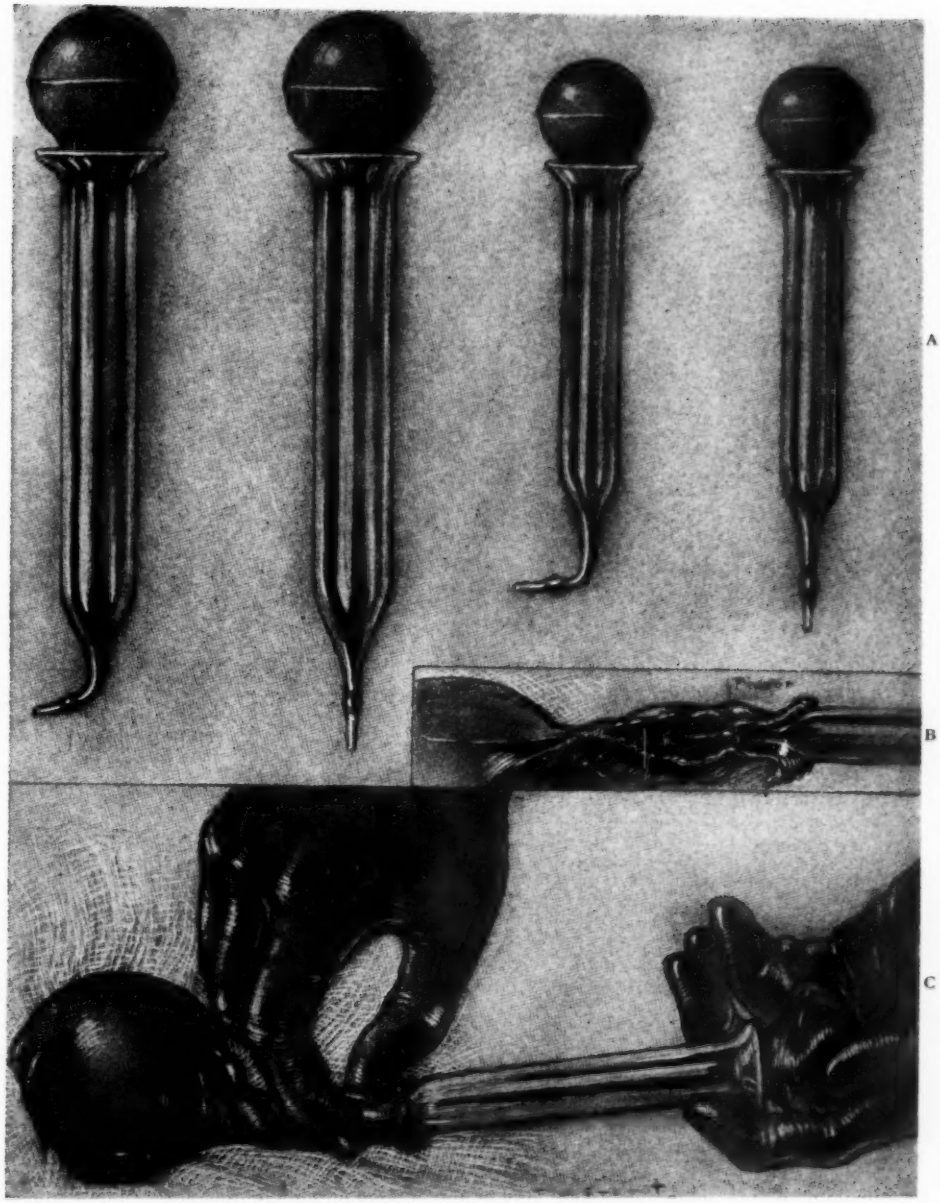

FIG. 110. A. Special type bulb syringe for distending tube. B and C. Tube held by fingers and air forced into the tube. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

obstruction. The tube is again tested with the syringe for patency. If patent, air bubbles can usually be seen at the cut end of the tube.

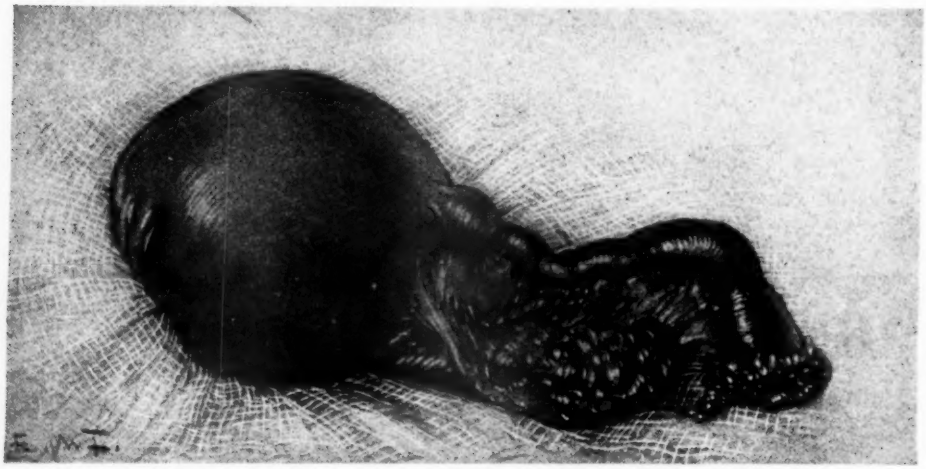

FIG. 111. Incision of tube just distal to the obstruction. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

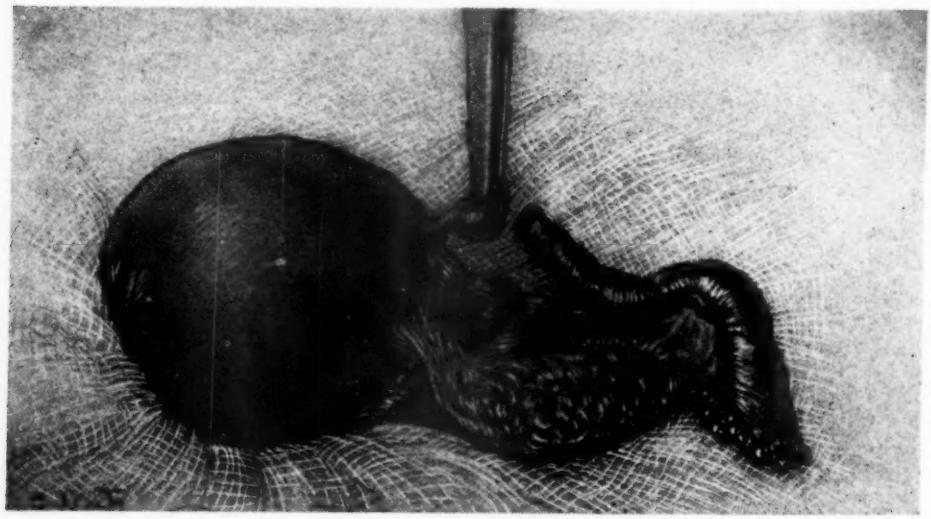

FIG. 112. Obstructed portion of tube excised; incision made close to the tube so as to avoid injury of vessels of the broad ligament. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

The proximal end of the tube, having been freed from the broad ligament, is now lying free except at its attachment to the uterine horn.

All bleeding points are very carefully clamped and ligated with very fine suture material.

Step 4. The removal of the uterine end of the tube and the

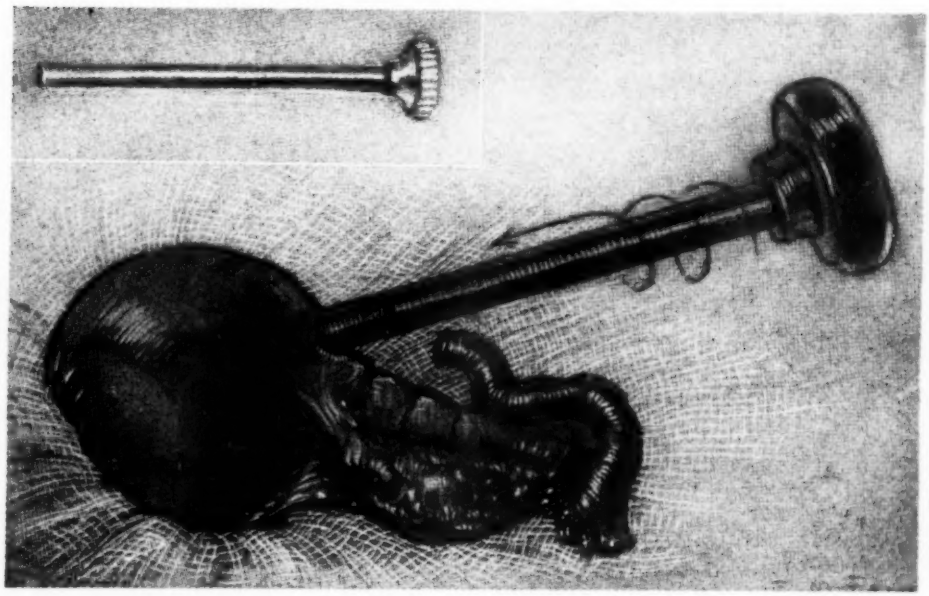

FIG. 113. A. Sovak Reamer. B. Reamer excising core from cornu of uterus. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

creation of a new opening into the uterine cavity is performed as one procedure. The uterus is held in the left hand (for removal of the right tube and in the right hand for removal of the left tube). The cut end of the tube is threaded into the reamer and with a circular motion a core is removed from the cornu of the uterus (Fig. 113B). When the reamer enters the uterine cavity there is a sudden "giving away" sensation. The piece of uterine horn and tube remains on the inside of the reamer. Thus an opening is created through the uterine wall and into the cavity of the uterus.

Step 5. In this step it is necessary to implant the patent cut end of the tube through the opening just created into the uterus. To do this it is necessary to strip a little of the tube from its mesosalpinx. The proximal end of the patent tube is cut from its mesosalpinx for a distance of about 0.5 cm. All bleeding points are ligated with fine ligatures. With a pair of

fine scissors this portion of the tube is cut on either side in its long diameter, thus creating a double edged tongue to the proximal end of the tube, alligator mouth (Fig. 114). The lumen of the tube is in the center of the wedge.

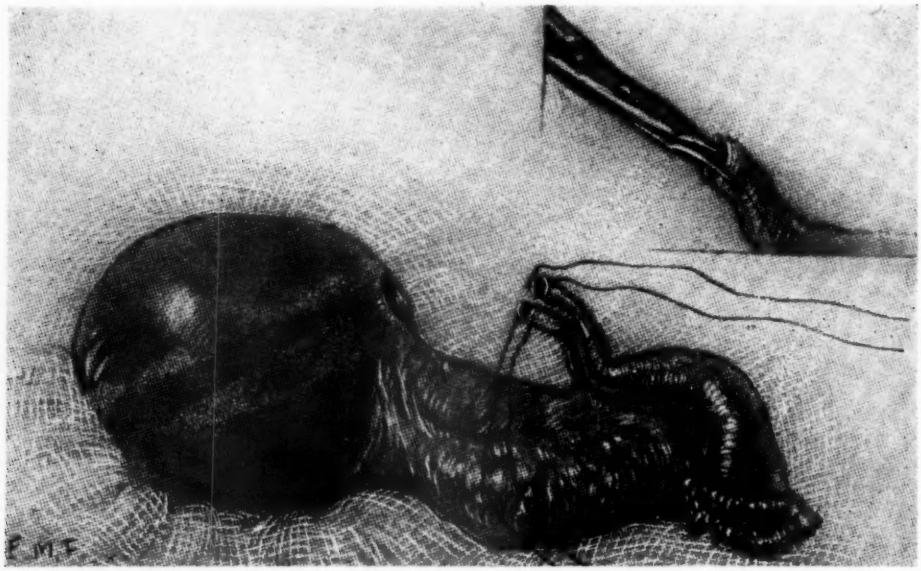

FIG. 114. Insert-tube cut on each side with fine scissors. Suture in each end of cut tube (alligator mouth). (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

Using a large Mayo needle threaded with fine chromic catgut No. 1 cut in two, pass this suture through the upper end of the wedged end of the tube. The same needle and catgut is then passed through the opening in the cornu of the uterus into the cavity of the uterus and is brought out through the fundus. The needle is removed and threaded on the other end of the suture. This is also introduced into the cavity through the same opening and is brought out through the fundus a short distance from the other suture. Both sutures are clamped and left long.

Another suture on a Mayo needle is then passed through the lower end of the tube, introduced into the uterine cavity through the same opening, but is brought out through the lateral wall of the uterus. The same procedure is carried out with the other end of the suture. A clamp is applied to the two ends of this suture.

When these two sutures are tied, the following occurs: (Fig. 115). The tube is drawn in and sutured to the wall of the uterus, opening the wedged end of the tube widely, leaving the

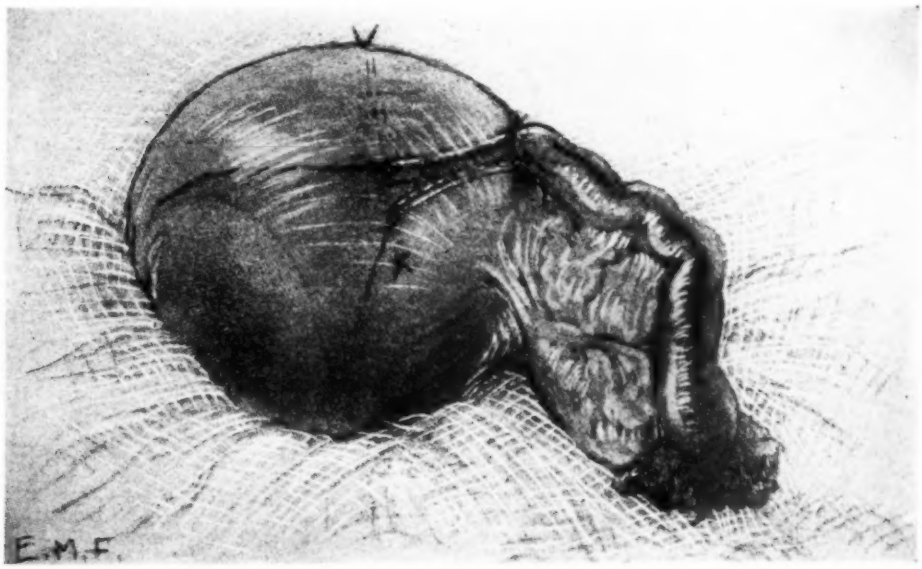

FIG. 115. Tube drawn through opening in the uterus and fixed to the wall of the uterus; opening the wedged end of the tube widely. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

proximal end of the tube patent. To test the patency of the reconstructed tube, air is again blown through with the syringe. If patent, bubbling will be felt in the cavity of the uterus.

The same procedure or another procedure if necessary may be carried out on the other side.

Step 6. The abdomen is closed in the usual manner. Before closing, however, a one-point suspension of the uterus should be performed.

In these cases no tubal insufflation should be done for three months postoperatively; after that it can be carried out. A sufficient time must be given for healing. The uterine end does not close as readily as the fimbriated end.

Question: Is the tube anchored to the outside of the uterus?

Answer: If the opening in the uterus is larger than the tube, close the opening with an extra stitch. As a rule, the opening

in the uterine wall should be large enough not to constrict the tube. It is not necessary to anchor the tube at the outer uterine wall.

Question: Is it necessary to always suture the ends of the tube to the fundus or anterior wall of the uterus?

Answer: No, they may be placed on the anterior and posterior wall, it makes no difference.

Question: Is there any objection to leaving a catheter in the lumen of the tube and out through the cervix?

Answer: Yes, this would be inviting adhesions and also inviting inflammatory conditions of the tube.

Question: When could pregnancy take place after that?

Answer: Cases have been reported with pregnancies within three months.

Question: Are these cases prospective cesarean sections?

Answer: Not necessarily; it depends entirely on the pelvis of the patient. If this operation is all that is done, there should be no danger in allowing the patient a test of labor.

PROCEDURE 2. CIRCUMCISION OF FIMBRIATED END OF TUBE FOR TUBES THAT ARE PATENT THROUGHOUT EXCEPT AT THE FIMBRIATED END; (SALPINGOSTOMY).

X-ray examination in these cases shows a tube patent throughout its entire course except at the fimbriated end. The roentgenogram will show a thin line of lipiodol going outward to the end of the tube and there it will form a little knob. In some instances, as hydrosalpinx with patent uterine end, the tube may be quite distended throughout.

Step 1. The abdomen is opened in the usual way. The intestines are walled off and all adhesions between the fimbriated end of the tube and the surrounding structures are cut with sharp dissection. Careful hemostasis is observed.

Step 2. The closed fimbriated end of the tube is delivered into the operative field and the distal end is cut across. It is advisable to cut it at an angle from above down. All bleeding points are very carefully clamped and ligated. A ureteral catheter is inserted into the fimbriated end of the tube. The

catheter is caught with a Bonney clamp through the tube, with the points of the clamp toward the fimbriated end of the tube (Fig. 116A). The patency of the tube is then tested by

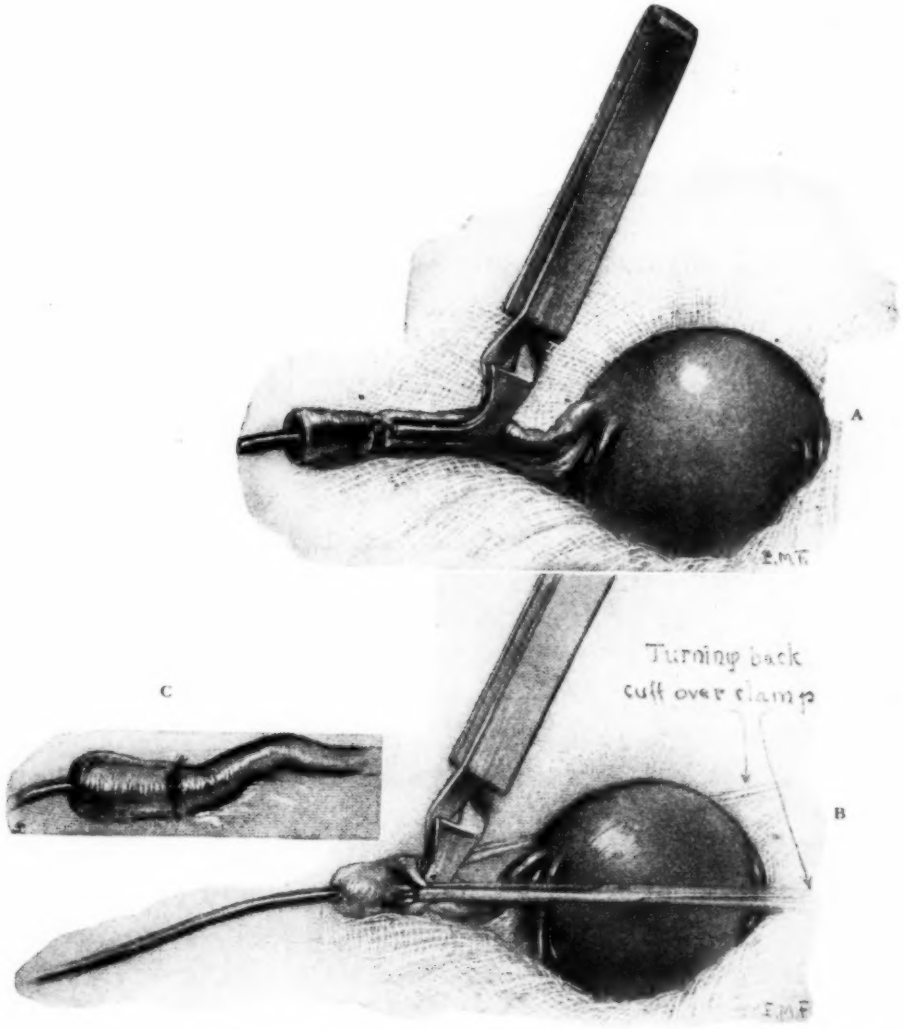

FIG. 116. A. Catheter caught by Bonney Clamp through tube; B. Distal end of tube brought over end of clamp and the lumen everted; C. Everted edge sutured, clamp removed, catheter in lumen of tube. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

blowing air through the catheter with the bulbous tipped syringe. If patent, one can feel air bubbling into the uterus. As

soon as the patency of the tube has been established the circumcision of the tube can be carried out.

Step 3. The distal end of the tube is brought over the end of the clamp and the lumen of the tube is everted (Fig. 116B) and is held there by several fine catgut stitches. The everted edge of the fimbriated end of the tube is sutured to the rest of the tube. The clamp is removed, the catheter is removed and there is now a patent fimbriated end of the tube, with the tube lumen presenting to the cavity of the peritoneum (Fig. 116C). The same procedure or another procedure if necessary is carried out on the opposite side.

Step 4. A one-point suspension is performed and the abdomen closed in layers.

If on *x*-ray examination with lipiodol the tube is found to be closed at the uterine end and on opening the abdomen the fimbriated end is found to be closed, the fimbriated end of the tube may be opened as in Procedure 2, the patency of the tube tested and if the tube is patent except at its uterine end Procedure 1 can be carried out on the uterine end and Procedure 2 may be performed on the fimbriated end.

In those cases where only a circumcision of the tube has been done, tubal insufflation must be begun within three days after the operation and every other day after that; otherwise adhesions will form at the fimbriated end of the tube and it will again be closed. After the patient leaves the hospital, the insufflation should be repeated every month for several months. Although this operation looks best, it probably is the least desirable. The number of pregnancies in these patients is about 2 per cent. In the tubal implantations, Procedure 1, the incidence of pregnancy in the literature is about 10 per cent.

Question: What are the chances of tubal pregnancies in these procedures?

Answer: The possibility of tubal pregnancy must always be considered in these patients. Tubal pathology was present in 90 per cent of 365 ectopics, seen on the gynecological service at Harlem Hospital.

PROCEDURE 3. ESTES OPERATION

This procedure is indicated:

a. When complete occlusion of the lumen of the tube is found;
b. When both tubes are pathological (pus tubes) and are removed;
c. When both tubes have been removed at a previous operation,

and the patient is very desirous of at least a chance of becoming pregnant.

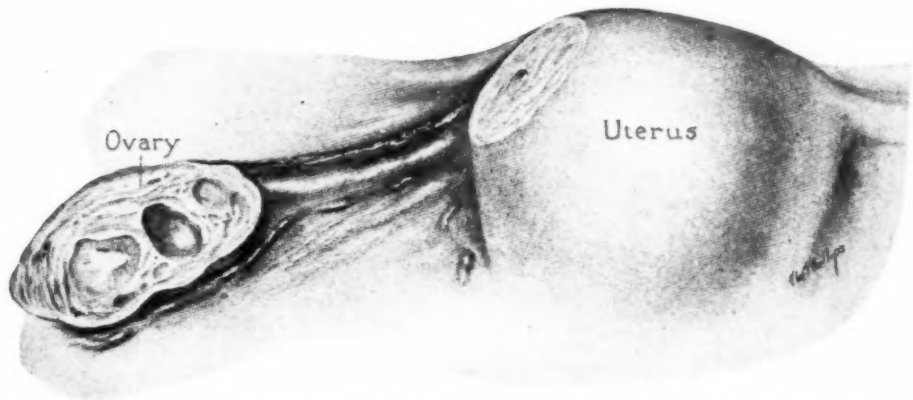

FIG. 117A. Uterine horn and ovary cut preparatory to transplantation. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

Step 1. The first step is the same as in the preceding operations. If the tubes have not been removed but are so pathological as to require excision, they must be removed in such a way as not to injure the blood supply to the ovary (p. 94). The broad ligament is cut very close to the tube and the fine clamps applied only to the bleeding points. Each individual bleeding point is ligated separately. This leaves the ovary suspended on a long mesosalpinx and the infundibulopelvic ligament.

Step 2. Preparation of the uterine cornu for the reception of the ovary. A piece of the uterine horn is removed by making a diagonal incision across the cornu of the uterus (Fig. 117A). This should leave an elliptical surface about as large as the

ovary. In the center of this surface an opening is made into the uterine cavity with the reamer (Fig. 117B). A thin piece is then removed from that part of the ovary which will allow

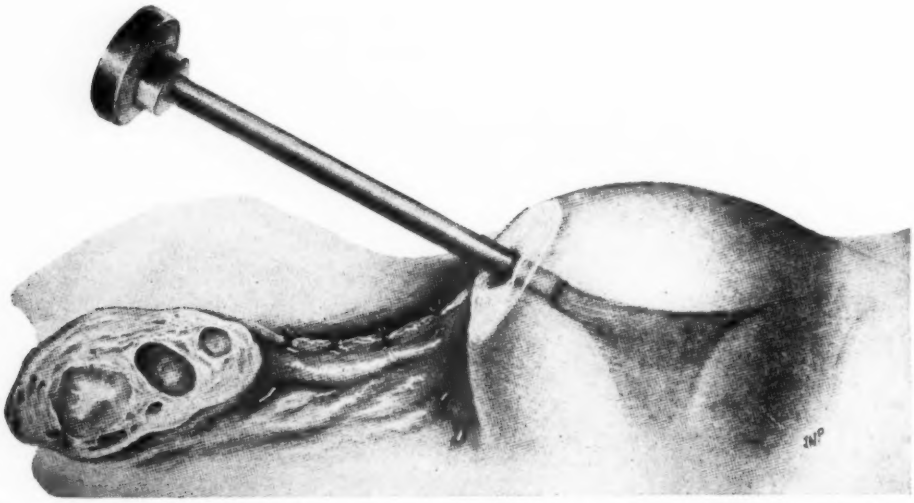

FIG. 117B. Opening into uterine cavity made with reamer. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

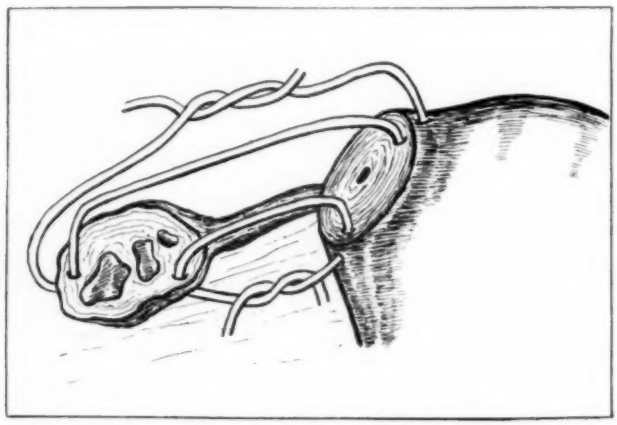

FIG. 118. Suture of the cut surface of the ovary to the raw surface of the uterus.

its approximation to the cornu of the uterus, without tension and without angulation to the blood supply from the infundibulopelvic ligament. The raw surface at the cornu of the

uterus and that on the ovary should be approximately the same size.

Step 3. A fine chromic catgut suture (No. 1 cut in three),

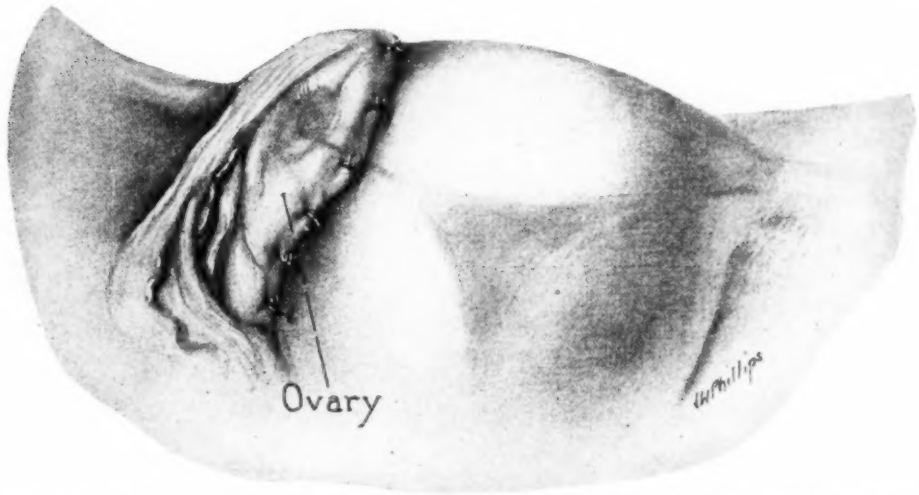

FIG. 119. Appearance of ovary and uterus after operation is completed. Uterine cavity communicating with ovary through cornu. (Sovak, F. W. *Am. J. Surg.*, 33: 406–421, 1936.)

is passed through the lowermost portion of the uterus and through the lowermost portion of the ovary (Fig. 118). This suture is clamped and left long. It is not tied until after the ovary is approximated with the next stitch. Another suture of the same material is passed through the distal portion of the ovary and the edge of the fundus of the uterus, so that when it is tied it will bring the raw surface of the ovary in contact with the raw surface of the uterus. Several chromic sutures are introduced along these cut edges of ovary and uterus holding them in approximation. The cavity of the uterus will thus communicate with the ovary through the canal of the cornu (Fig. 119). It is hoped that this canal will ultimately become lined with endometrium or possibly some form of epithelium from the ovary itself. It is also hoped that ovulation will take place directly into this opening thus allowing impregnation to take place in the uterine cavity. Some men have transplanted the ovary directly into the uterine cavity (Tuffier,

Morris). This hardly seems necessary since an ample opening can be made with the reamer.

Step 4. The same or some other form of surgery may be necessary on the opposite ovary.

Step 5. The abdomen is closed in the usual manner.

[*To be continued in subsequent issues.*]